Ontario

Report
of
The Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario

Volume One

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Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario

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April 1984

May it please Your Honour:

By Order-in-Council 1243/80, dated the 29th day of April, 1980, we were constituted a Royal Commission under the Public Inquiries Act to inquire into matters of health and safety arising from the use of asbestos in Ontario. We have completed our inquiry and beg to submit the accompanying Report.

J. Stefan Dupré
J. Stefan Dupré
Chairman

J. Fraser Mustard
Commissioner

Robert J. Uffen
Robert J. Uffen
Commissioner

Order-in-Council

O.C. 1243/80

Copy of an Order-in-Council approved by Her Honour the Lieutenant Governor, dated the 29th day of April, A.D. 1980.

The Committee of Council have had under consideration the report of the Honourable the Minister of Labour wherein he states that

WHEREAS, the Government of Ontario believes that it is in the public interest that ample opportunity is afforded for full public input into the assessment of the matter of health and safety arising from the use of asbestos in Ontario and that the benefit of the best medical, scientific and other expert advice, both from within the government and from the community at large is made available in arriving at conclusions as to the need for further remedial action, and

WHEREAS, the Government has reached the conclusion that these aims can best be achieved by means of a public inquiry instituted pursuant to the provisions of The Public Inquiries Act, 1971, Chapter 49;

The Honourable the Minister of Labour therefore recommends that a Commission be established pursuant to the provisions of The Public Inquiries Act, 1971, Chapter 49, to study and report to the Minister of Labour on the matter of health and safety arising from the use of asbestos in Ontario and:

1. To investigate all matters relating to health and safety arising from the use of asbestos in Ontario;

2. To identify the relevant data related to asbestosis, mesothelioma and other diseases and health hazards of persons working with or exposed to asbestos in Ontario;

3. To review the present basis for Workmen's Compensation Board awards as they relate to occupational health matters affecting workers exposed to asbestos, including any special programs dealing with the rehabilitation of such workers;

4. To make such recommendations in relation to the above as the Commission deems appropriate.

The Honourable the Minister of Labour further recommends that the following persons be appointed members of the Commission:

Dr. Stefan Dupré
Dr. Fraser Mustard
Dr. Robert Uffen

and that Dr. Stefan Dupré be designated Chairman of the Commission.

And the Honourable the Minister of Labour further recommends that all government ministries, boards, agencies and commissions assist the commission to the fullest extent in order that it may carry out its duties and functions,

AND THAT the commission have authority to engage counsel, expert technical advisors, investigators and other staff as it deems proper at rates of remuneration and reimbursement to be approved by the Management Board of Cabinet in order that a complete and comprehensive report may be prepared and submitted to the Government,

AND THAT the Ministry of the Attorney General will be responsible for providing administrative support to the commission.

The Committee of Council concur in the recommendations of the Honourable the Minister of Labour and advise that the same be acted on.

Certified,

(signed) Deputy Clerk, Executive Council

Acknowledgements

A report of this size and complexity is by necessity the product of many hands. As Commissioners, we alone are responsible for its content, but our indebtedness for its existence encompasses many individuals.

The three members of our senior staff, Dr. Donald N. Dewees, Director of Research; Mr. John I. Laskin, Legal Counsel; and Miss Linda B. Kahn, Executive Co-ordinator, were to us full partners in every sense save responsibility for the end product. Their selfless devotion and dedicated professionalism infused every stage of our work. As the only partner who was engaged in this enterprise on a full-time basis, Miss Kahn deserves special gratitude for orchestrating us all and for bearing enormous administrative and editorial burdens with gracious aplomb and awesome efficiency.

We gratefully acknowledge the devoted services of the members of our research, administrative, and secretarial staff, whose names appear in Appendix G at the end of this Report. In particular, we thank Mrs. Phyllis D. Bartley, who served simultaneously as Miss Kahn's right hand and as our senior secretary from the day we were appointed. To our all-purpose researcher, Mr. Ronald J. Daniels, and our librarian, Mrs. Patricia S. Rubin, we also express special gratitude.

For the knowledge that infused our deliberations, we owe an enormous debt to the Canadian, American, British, and Swedish scientists and officials who appeared before us as expert witnesses (see Appendix B); to the authors and reviewers of our research studies (see Appendix F); and to our consultants (see Appendix H). Among the latter, we wish to cite the especially generous contribution in time and effort of Professors David C.F. Muir, Robin S. Roberts, and Neil Rowlands.

Throughout the many days on which we heard sworn testimony, we drew invaluable assistance from the individuals who acted as the representatives of the several parties granted formal standing by this Commission

(see Appendix A). Their examination and cross-examination of witnesses added materially to the quality of the evidence before us. We express our gratitude to these representatives, Ms. Linda Jolley and Messrs. Jean Bazin, Orlando Buonastella, Phillipe Casgrain, Ed Cauchi, Rick Evans, Brian Gibson, Timothy S. Hardy, Thomas R. Lederer, Nick McCombie, James McNamee, Arthur F. Sampson III, David K.L. Starkman, Daniel Ublansky, and Edward W. Warren.

Finally, we warmly thank the many persons who appeared before us during the informal phases of our hearings. The extent of their assistance is visibly apparent in the text of our Report and is cited in its documentation.

J.S.D.
J.F.M.
R.J.U.

Part I

Introduction

Chapter 1 The Report of The Royal Commission on Asbestos: An Overview

A. Another Royal Commission

Was this Royal Commission really necessary?

Perhaps deservedly, perhaps not, royal commissions in Canada have acquired a reputation for being slow, elaborate, and expensive means of investigating subjects of varying degrees of public importance and controversy. The range of these subjects has been almost infinite: they have been as specific as the affairs of a lone person and as vast as the economic prospects of the entire realm. We were appointed by the Government of Ontario to deal with matters of health and safety arising from the use of a single substance in this province: asbestos. That asbestos can be a devastatingly serious health hazard was widely known many years before this Commission was appointed in April of 1980. We were hardly needed to confirm this fact.

This stark fact has understandably aroused widespread public apprehension. Asbestos is everywhere: it is in the air we breathe, the water we drink. It has been used for generations because of its superb fire- and acid-resistant qualities. Canada is the largest producer of asbestos in the western world. World-wide, asbestos has brought disease and death to literally thousands of individuals who have been exposed to its microscopic fibres as miners and millers, manufacturing workers, insulators, and members of other occupations. It turns out that these individuals, as conveyed by the title of a well-known book on the subject, were literally *Dying for a Living*.[1] What are the implications for the continued use, if any, of asbestos in industry? More generally, does this known workplace killer threaten unsuspecting individuals in office buildings, airports, schools?

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[1] Lloyd Tataryn, *Dying for a Living* (Toronto: Deneau and Greenberg Publishers Ltd., 1979).

Necessary or otherwise, this Commission had no shortage of attention. An initial public meeting, convened in October of 1980, attracted about 400 individuals, and this attendance was almost matched at a second public meeting six weeks later. These meetings had been announced in advertisements placed in 81 daily and weekly newspapers throughout Ontario. The advertisements also publicized the existence of a brochure which was available on request and which described our plan of activities. This brochure, which was also mailed on an unsolicited basis to persons likely to have an interest in our terms of reference, included a return postcard on which the recipient could indicate the desire to be placed on the Commission's mailing list or declare the intention to make a written or oral submission. Over 2,000 individuals and organizations placed themselves on our mailing list and were thereby kept abreast of all hearing dates and of our research programme. During the week of February 16 through 20, 1981, we sat morning, afternoon, and evening hearing oral submissions in Toronto in a setting as informal as the intermittent glare of television lights permitted. The presenters ranged from seriously ill workers and lone widows to leaders of major business, labour, and government organizations. Other such informal hearings ensued, in Windsor on March 27, 1981 and Toronto on June 8, 1981. But royal commissions, at least in Ontario, involve more than informal hearings; this is perhaps not least of the reasons why they are reputedly slow and ponderous.

Pursuant to section 5(1) of the *Public Inquiries Act,*

> A commission shall accord to any person who satisfies it that he has a substantial and direct interest in the subject-matter of its inquiry an opportunity during the inquiry to give evidence and to call and examine or to cross-examine witnesses personally or by his counsel on evidence relevant to his interest.[2]

In October of 1980, we gave notice that persons who wished to apply for standing pursuant to section 5(1) should so indicate by January 31, 1981, and we thereupon granted standing to the twenty parties listed in Appendix A at the end of this Report. We decided further that one of these parties, the Asbestos Victims of Ontario, should be granted public monies to defray the costs of its representation. The Asbestos Victims of Ontario is a group composed of former asbestos workers, their spouses, or widows.

Over the ensuing months, the several representatives of the parties-with-standing met regularly with our legal counsel to develop, by consensus, the names of witnesses who should be invited to testify. The response rate to our invitations turned out to be almost 100%. The names of our witnesses are listed in Appendix B. These 53 men and women came to give sworn testimony not only from Ontario and elsewhere in Canada but from Sweden, the United Kingdom, and the United States. Most underwent one and sometimes two full days of examination and cross-examination. About half our witnesses were scientists, constituting an international who's who of experts on the health effects of asbestos. The taking of formal testimony consumed 53 hearing days, concentrated mainly during the summer months of 1981 and 1982.

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[2] R.S.O. 1980, c. 411.

While our hearings were in progress, we undertook, either personally or through our staff, to acquire yet additional knowledge through direct observations and informal contacts. This involved, by way of illustration, visits to some Quebec asbestos mines, to the Mount Sinai School of Medicine in New York, to certain Ontario manufacturing establishments, and to some asbestos removal projects. We also undertook a research programme to improve our understanding of the issues before us. This programme yielded two background papers and ten book-length research studies, listed in Appendix F, which we published and released for assessment and criticism as they were completed. The subjects covered by these studies ranged from the technical problems of measuring asbestos levels to the matter of workers' compensation for asbestos-related disease in Ontario. Because the studies began to appear in February of 1982, the information they divulged and the perspectives they provided became a useful adjunct to our remaining hearings. Our last day of formal hearings was August 24, 1982. After allowing the parties-with-standing sufficient time to review the evidence, we returned to the hearing room for one final occasion: the presentation of final submissions by these parties on January 28, 1983. Otherwise, we sat closeted in deliberations with our director of research, our legal counsel, and our executive co-ordinator, surrounded by our 12 research studies and background papers, our 88 written submissions, and our 8,378 pages of oral presentations and sworn testimony.

This Report is the outcome of those deliberations. Both its length and its prose — a literary genre we call "commissionese" — ensure that it will be read from cover-to-cover by precious few. In this chapter, we shall attempt to distil our findings to their bare essentials. Readers who — from anger or delight, bemusement or curiosity, or any other motive — desire elaboration need only glance at the footnotes to find the particular chapters or sections of chapters that divulge our information base and our detailed reasoning. We have been prescriptive as well as analytical; we shall therefore convey the gist of our prescriptions as this chapter unfolds and provide, at its end, the text of each of the 117 formal recommendations that appear in the body of the Report.

B. Health Effects of Asbestos

Residents of Ontario have more than the normal run of reasons to share the international feelings of apprehension that asbestos has aroused. This province is the scene of what we document in this Report to be a world-class occupational health disaster: the Johns-Manville plant in Scarborough, in the Municipality of Metropolitan Toronto. This plant, between 1948 and 1980, manufactured asbestos-cement pipe, using a mixture of two kinds of asbestos: chrysotile and crocidolite. At various times during its existence, this plant also manufactured asbestos-cement board, using only chrysotile, and asbestos insulation materials, using chrysotile and a third kind of asbestos called amosite.[3]

As of 1983, the death toll from asbestos exposure in this plant, as measured by the number of claims awarded by the Ontario Workers' Compensation Board, was 68.[4] This lone plant, whose annual employment never exceeded 714 workers, has already occasioned more deaths from industrial disease than the entire Ontario mining industry, which annually employs over 30,000 workers, occasions from industrial accidents in an average four-year period.

The death toll at this plant, which closed in 1980, offers harsh testimony to the nature of long-latency disease.[5] It has mounted gradually and inexorably because individual deaths are separated from the beginning of the exposures that caused them by some 10 to 30 or more years. The dimensions of the disaster have therefore been growing over time; for example, between August 1981 and August 1982, the middle year in the existence of this Commission, 5 more ex-employees died of mesothelioma, a rare cancer that is specifically associated with asbestos exposure. It is a tragically safe assumption that, among those who worked in this plant, asbestos-related deaths will continue to occur, and that hence the disaster has yet to run its course.

Asbestos-related deaths are from mesothelioma. They are from lung, gastrointestinal, and laryngeal cancers which, while they arise from a variety of sources, can also be caused by asbestos. They are from asbestosis. The last of these diseases, as its name suggests, is asbestos-specific. It is a form of lung fibrosis which can itself be fatal, but more commonly makes its victims susceptible to death from related causes and, most significantly, is an irreversible, disabling, normally progressive condition. By the time the Johns-Manville plant closed in 1980, 113 of its exposed workers had been awarded disability pensions by the Workers' Compensation Board for their varying degrees of medical impairment occasioned by asbestosis.

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[3] For a description of the different types of asbestos, see Chapter 2, Section B.

[4] For a full account of the disease and mortality experience in this plant, see Chapter 3, Section A. For an account of the cost of compensating the plant's victims, see Chapter 14, Section C. A special rehabilitation effort undertaken for workers at this plant by the Workers' Compensation Board is discussed in Chapter 14, Section A.

[5] For a description of asbestos-related diseases and the phenomenon of long latency, see Chapter 2, Section D. For a further discussion of the time interval between the initial exposure to asbestos and the clinical manifestation of disease, see Chapter 5, Section E.4.

The asbestos-induced disaster at this plant ranks with the worst that have been recorded in the international epidemiological literature on asbestos. It places the name Scarborough on an unenviable list with Charleston, South Carolina; Rochdale, England; and a handful of other places.[6] The Scarborough plant accounts for half of all the asbestos deaths and disabilities that have been compensated by the Ontario Workers' Compensation Board. The remainder have been occasioned by exposure in a wide variety of work situations and industrial processes, for example, wartime gas mask manufacturing, brake manufacturing, and shipbuilding. The Ontario employers whose workers suffered asbestos disease and death are spread throughout the province; excluding Johns-Manville, only two have given rise to more than 5 awarded claims.[7]

There is indeed reason to be apprehensive about asbestos in Ontario. But what is this Commission's "bottom-line" on the nature of the health hazard posed by asbestos? Our "bottom-line" can be reduced to the following propositions:

1. Asbestos diseases offer a classic instance of the phenomenon that is called a dose-response relationship. The greater the exposure of an individual to asbestos (the dose), the greater the likelihood of disease (the response). Conversely, the lower the exposure of an individual to asbestos, the lower the likelihood of disease.[8] However, at low levels of exposure, the assessment of disease risk is bedevilled by uncertainty. The uncertainty arises from a number of factors including difficulties of measurement, limitations of epidemiology, and lack of data.[9] Accordingly, prudence dictates the assumption that any exposure involves some risk of disease.

2. The overwhelming weight of the scientific evidence is that disease results from breathing (inhaling) asbestos fibres. There is no substantial evidence that disease can result from swallowing (ingesting) asbestos fibres

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[6] For a comparative analysis of disease and death among asbestos workers in various plants throughout the world, see Chapter 5, Section B.

[7] For an account of the volume of compensated disease and death claims among various Ontario employers, see Chapter 3, Section A.

[8] For a discussion of dose-response relationships for the asbestos-related diseases, see Chapter 5, Section E and Chapter 7, Section C.

[9] For a discussion of these factors, see Chapter 4, Section D.

and only the most speculative of hypotheses on how ingestion could give rise to disease.[10]

3. The asbestos fibres which are most likely to cause adverse health effects when inhaled are long and thin. "Length" and "diameter" are, of course, relative phenomena: fibres are measured in microns, one micron being one-millionth of a metre. The hazardous asbestos fibres are those which would be longer than 5, perhaps longer than 8 microns, and thinner than 1.5 or perhaps 0.25 microns.[11]

4. There is strong evidence that crocidolite and amosite fibres tend to be more hazardous than chrysotile fibres, primarily because they are more likely to conform to the most hazardous length and diameter and secondarily because they are more likely to become airborne and hence to be respirable. It is also possible that crocidolite and amosite are more hazardous because of their chemical composition, but this possibility rests in the realm of speculation.[12]

5. All fibre types can cause all asbestos-related diseases, but mesothelioma is most likely to result from crocidolite exposure, has a strong association with amosite exposure, and has a weak association with chrysotile exposure.[13]

6. Whatever their type, the likelihood that asbestos fibres of the most hazardous dimensions are respirable is strongly a function of the individual process in which asbestos is being used. Thus, for example, the manufacture of brake linings, which involves drilling and grinding, is much less likely to generate fibres of hazardous dimensions than textile manufacturing, which involves spinning and weaving.[14]

7. The individuals who have been afflicted with asbestos-specific diseases have almost invariably been occupationally exposed to asbestos. However, there is incontrovertible evidence that mesothelioma has afflicted individuals who shared the domicile of an asbestos worker at the time this

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[10] For an account of the biological evidence as to the ingestion of asbestos fibres, see Chapter 4, Section B.2. For a discussion of the epidemiological evidence, see Chapter 5, Section I.

[11] For an analysis of the importance of fibre dimension, see Chapter 4, Section B and Chapter 5, Section D.

[12] For a discussion of the evidence which indicates that crocidolite and amosite tend to be more hazardous than chrysotile, see Chapter 5, Section C. For an account of the significance of fibre dimension and the possible significance of fibre chemistry, see Chapter 5, Section D.

[13] For an account and an analysis of the evidence concerning the incidence of mesothelioma in relation to different fibre types, see Chapter 5, Sections C.4, C.6, and D; and Chapter 7, Sections C and F.

[14] For a review of the health experience of workers in various processes using asbestos, see Chapter 5, Section B and Chapter 7, Section C. For a discussion of the effects of process on fibre dimension and the consequent health effects, see Chapter 5, Section D and Chapter 7, Sections C and F. For a description of different industrial processes, see Chapter 6, Section C.

worker was exposed. There is weak evidence that individuals may have contracted disease from exposure to asbestos in the neighbourhood of asbestos plants.[15] We have found no evidence that disease afflicts individuals who breathe asbestos in the outdoor air or inhale it as occupants of asbestos-containing buildings.[16]

8. Asbestosis is a disease which is attributable solely to occupational exposure to asbestos. There is substantial evidence that at a very low level of occupational exposure to asbestos, the fibrotic process in the lungs, if indeed it can even be initiated, will not develop to the point of producing asbestosis at the level of clinical diagnosis or of producing any manifestation that would cause even the mildest discomfort to an individual.[17]

9. There exists no relationship between smoking and mesothelioma. Asbestosis cannot be caused by smoking, but there is evidence that smoking can accelerate the pace at which the fibrotic process develops. Finally, while asbestos alone can cause lung cancer, there is clear evidence that asbestos and smoking interact so synergistically in causing this disease that smoking coupled with asbestos work is like pouring gasoline on a fire.[18]

We have made a number of other findings on the health effects of asbestos, but the above can be taken to be the highlights. What are the implications of these findings? We answer this question first by examining asbestos in fixed place industry, then asbestos in buildings, and finally asbestos elsewhere.

C. Asbestos in Fixed Workplaces

Fixed workplaces, which in the case of asbestos mean mining and manufacturing, involve, for regulatory purposes, a distinct compartment of asbestos-related activities for several reasons. First, these activities are optional; they could be banned if asbestos exposure were deemed to involve unacceptable risks. Second, these activities, being confined to fixed workplaces, are amenable to work practices and engineering controls which reduce the concentration of asbestos fibres to which workers are exposed. Third, and again because these activities are in fixed workplaces, it is possible to monitor, in an ongoing manner, the concentration of asbestos fibres in the air. The prevailing measurement technique involves: (i) air sampling by what is known as the membrane filter method; (ii) counting the number of fibres captured from each air sample with the use of an optical microscope; and (iii) expressing the number of fibres detected on the basis of so many fibres per cubic centimetre of air [fibres per cubic centimetre (f/cc)].[19]

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[15] For an elaboration of these points, see Chapter 5, Section E.

[16] See Chapter 11, Section C and Chapter 9.

[17] For a discussion of this evidence, see Chapter 5, Section E.3.

[18] For a discussion of the relationship between smoking, asbestos exposure, and disease, see Chapter 5, Section F.

In accordance with the provisions of the Ontario *Occupational Health and Safety Act*, asbestos is a designated substance and is covered by the Regulation Respecting Asbestos. This Regulation enjoins fixed place employers to "take all necessary measures and procedures" to ensure that the exposure of a worker to airborne asbestos "is reduced to the lowest practical level" and stipulates control limits which are not to be exceeded. These limits are 1 f/cc for chrysotile, 0.5 f/cc for amosite, and 0.2 f/cc for crocidolite. The Regulation extends to all fixed place, asbestos-using industry and does not discriminate among industrial activities.

Historically in Ontario, asbestos has been mined and used in a wide range of manufacturing processes. Mining, which involves chrysotile asbestos, is for the time being a trivial to non-existent activity; manufacturing, to the best of our knowledge, is currently confined to the use of chrysotile asbestos and is in the main devoted to friction products, including automotive brakes.[20]

What is the risk to which workers are exposed from these and, for that matter, any other asbestos-using activities that involve fixed place industry? Asbestos is one of the most thoroughly studied industrial carcinogens in the world. There accordingly exists a data base from which to estimate the risk of disease at different levels of occupational exposure. We have used models of disease incidence and data from a number of epidemiological studies of workers exposed to asbestos to forecast the possible disease risk that may result from exposure to varying concentrations of all types of asbestos dust in varied manufacturing processes.[21]

On the basis of this analysis, we conclude that we cannot condone any manufacturing activity that involves the use of crocidolite or amosite asbestos.[22] The use of these two types of asbestos in Ontario should, therefore, be prohibited indefinitely.[23] As for chrysotile, it is necessary to distinguish the industrial processes in which asbestos is being used. The control limit that the Regulation Respecting Asbestos currently applies to all chrysotile processes is 1 f/cc. If this control limit is rigorously observed so that, at the level of an 8-hour time-weighted average, it is rarely exceeded, we calculate that the average level of exposure to which workers will be subjected is no greater than 0.5 f/cc.[24] Our data base permits us to calculate the disease risk encountered by workers in the following industrial processes: mining and milling; general manufacturing, excluding textiles and cement products; textile manufacturing; and cement product manufacturing. We find in the cases of chrysotile mining and milling and of general chrysotile manufacturing that the disease risk associated with chrysotile exposure under a 1 f/cc control limit, effectively enforced, involves a projected mortality rate well below the mortality rate that results from industrial accidents in all Ontario manufacturing. It therefore falls well within the bounds of a societally acceptable industrial risk.[25]

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[19] For a detailed description and analysis of the membrane filter method, see Chapter 7, Section B.

[20] For a description of asbestos mining in Ontario, see Chapter 6, Section F; manufacturing activity is described in Chapter 6, Section B.

[21] These estimates are developed in Chapter 7, Section C. A technical description of the risk assessment model is contained in the Appendix to Chapter 7.

[22] For the criteria we have developed to assess when a substance should be subject to control limits, when it should be prohibited, and the relationship of a substance to the availability of substitutes, see Chapter 7, Section E. The feasibility and cost of asbestos control methods are discussed in Chapter 6, Section C. For a discussion of substitutes and their health effects, see Chapter 6, Sections D and E.

Textile manufacturing with chrysotile is starkly different. Under a 1 f/cc control limit, effectively enforced, the estimated risk from mortality in this asbestos-using process exceeds the risk of mortality from accidents in manufacturing and approaches or passes the risk of mortality from fatal accidents in general Ontario mining activity. This activity, with respect to industrial accidents, has long been one of the most hazardous in Ontario. To reduce the risk of death from disease in chrysotile-using textile product manufacturing to that in other chrysotile-using processes would require a control limit of 0.04 f/cc, far lower than any limit which, given prevailing measurement technology, could be enforced. We find that this chrysotile-using process therefore cannot be condoned in Ontario, and that it should be banned until it were to become possible to satisfy the Ministry of Labour that its introduction would involve risk of no greater significance than that posed by mining and general chrysotile manufacturing.

The risk level we associate with chrysotile mining and general manufacturing may be lower than the accident risk in manufacturing, but it is still significant and is a disease risk borne by asbestos workers who, like all manufacturing employees, run the risk of fatal industrial accidents. We, therefore, view the following as essential if chrysotile use is to be tolerated at a control limit of 1 f/cc: (i) All workers must be fully informed of the risk they are facing, as the Regulation Respecting Asbestos contemplates. (ii) The Regulation must be strictly enforced so that the control limit of 1 f/cc in fact ensures that actual average exposure will be 0.5 f/cc. (iii) The Regulation, the *Occupational Health and Safety Act*, and the practices and organization of the Ministry of Labour must all be modified to give assurance to the Government and Legislature, the public, and above all the workers concerned that the regulation of chrysotile asbestos is indeed being implemented effectively.[26]

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[23] See Chapter 7, Section F.3. The ban we recommend takes the form of outright prohibition by the Ministry of Labour which could only be lifted if health risks were greatly reduced. In the case of amosite, this would necessitate a currently unattainable exposure maximum of 0.1 f/cc. (See Recommendation 7.14.) In the case of crocidolite, the exposure maximum, if any, would have to be 0.02 f/cc. (See Recommendations 7.12 and 7.13.)

[24] For the basis of this calculation, together with an extensive discussion of sampling and measurement errors, sampling frequency, and related issues, see Chapter 7, Section B.

[25] For an extensive discussion of this point and of the points summarized in the balance of this paragraph and the following paragraph, see Chapter 7, Section F.

We have made formal recommendations in this regard which range from developing more representative air sampling procedures to increasing worker participation in the enforcement and monitoring process. We single out one of these recommendations, which is that the Ministry of Labour should create a Designated Substances Enforcement Unit to supplement the enforcement role of its inspectorate in workplaces where designated substances are present. Other than for its Director, whose qualifications would include training in investigative techniques, the Unit would not require the engagement of new Ministry staff but would instead involve selecting interdisciplinary teams of professionals from the Occupational Health Branch, assembled by the Director, for the purpose of making unannounced visits to plants using designated substances.[27]

D. Asbestos in Buildings

In Ontario, as in a number of other jurisdictions, asbestos was widely used until 1973 as sprayed insulation material for fire protection and acoustical purposes and in pipe and boiler insulation.[28] Accordingly, it is present in a large number of multi-storey buildings, including offices, schools, factories, and air terminals. The types of asbestos that are generally present are not only chrysotile but amosite.

Understandably, the association of chrysotile and amosite asbestos with buildings has created enormous public apprehension. Is this apprehension warranted? Our response to this question comes in two parts. First, asbestos is indeed a potentially serious hazard to workers who are directly engaged in such construction-related activities as asbestos removal and building demolition, or who are involved in a wide variety of activities in the realm of building maintenance.[29] Second, the inhalation of chrysotile and amosite fibres by building occupants almost never poses a health hazard, save perhaps if particularly elevated exposure is occasioned by the disturbance of asbestos, especially in removal projects.[30]

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[26] For an extensive discussion of the Regulation Respecting Asbestos, the internal responsibility system, and enforcement, see Chapter 8.

[27] See Chapter 8, Section E.1.

[28] For a discussion of the application and characteristics of sprayed insulation and pipe and boiler insulation, the buildings in which these are found and the types of asbestos used, see Chapter 9, Section B.

Let us immediately elaborate on our second finding. It is essentially in line with one made by the United Kingdom Advisory Committee on Asbestos in 1979. Notwithstanding this British finding, it seems evident that public apprehension about the risk which asbestos poses to building occupants is not easily allayed. If this is so, it speaks strongly in favour of more pointed and co-ordinated communication by public agencies. In particular, we find that a major cause of public apprehension must be attributed to the failure of government agencies involved in asbestos regulation to communicate clearly with respect to the all-important factor of measured levels of asbestos in building air. Ontario is itself a classic example of a jurisdiction in which two different agencies have utterly failed in co-ordinating their communication. Thus, in Ontario, the Ministry of Labour has promulgated well-publicized control limits for fixed workplaces. As we have seen, the chrysotile control limit is 1 f/cc. Meantime, the Ministry of the Environment has a guideline for asbestos in the ambient air (generally extrapolated to building air) of 0.04 f/cc. At first blush, it would appear that the Environment guideline is 1/25 that of the Labour control limit. But this is not true. The Labour control limit of 1 f/cc applies in a setting where the number of fibres in air samples is counted with the use of an optical microscope. By contrast, the low fibre concentration envisioned by the Environment guideline involves fibre counting by electron microscopy.[31] The electron microscope detects a great many fibres too thin to be seen through the optical microscope. Accordingly, a count of asbestos fibres in building air using electron microscopy will find more fibres, to the extent of an order of magnitude or perhaps 10 times as many fibres as a count through the optical microscope.[32] It follows that the Environment guideline of 0.04 f/cc has the effect of being less like 1/25 and more like 1/250 the Labour control limit.

We find that outdoor airborne asbestos fibre concentrations are often less than 0.001 f/cc measured using an electron microscope, and hardly ever reach 0.01 f/cc.[33] The optical equivalent of these concentrations would be an order of magnitude smaller. Perhaps more important, we conclude that the air in buildings with sprayed asbestos-containing insulation usually averages less than the equivalent of an optical fibre count of 0.001 f/cc, and the highest readings would rarely exceed 0.01 f/cc. Acknowledging that building air is contaminated not only by chrysotile but by the more hazardous amosite fibres, it is possible to estimate the risk to building occupants who are exposed to a mixture of chrysotile and amosite fibres at the level of 0.001 f/cc. The risk that an individual who had been exposed to asbestos in a building for, say, 10 years would die of an asbestos-related disease is less than 1/50 the risk that death would come from commuting 10 miles by auto to and from that building daily for the same period. At this level, we deem the risk which asbestos poses to building occupants to be insignificant and therefore find that asbestos in building air will almost never pose a health hazard to building occupants.[34]

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[29] For a discussion of the asbestos exposures to which building workers have been and can potentially be subjected, see Chapter 9, Sections B and D.

[30] For an analysis of the exposures of building occupants to asbestos, see Chapter 9, Section D.

[31] For a discussion of the ambient air guideline, see Chapter 11, Sections C.4 and C.5.

[32] For a discussion of the relationship between optical fibre counts and electron microscope fibre counts, see Chapter 7, Section B and Chapter 11, Section C.1.

[33] For a discussion of data on outdoor asbestos concentrations, see Chapter 11, Section C.2.

The same, however, is not true with respect to workers engaged in demolition, asbestos removal, or building maintenance activities. Such workers may become exposed to asbestos levels that greatly exceed the control limits which apply in fixed place industry.[35] The exposure involved is to amosite as well as chrysotile asbestos. This exposure, in turn, may come in short, intense bursts that can overcome the normal defence mechanisms of the human lung. Where demolition, removal, and certain repair activities are involved, the workplace is not fixed but instead resembles that of the typical construction project. It follows that worker protection can better be enforced by specifying work procedures than by using air monitoring to enforce control limits, and we prescribe elaborately in this regard.[36] The magnitude of the potential risk from exposure in building demolition is such that we specifically prescribe that all friable asbestos should be removed from buildings prior to demolition.[37]

Since 1979, numerous Ontario school boards, with some $26 million in financial assistance from the provincial government, have made massive efforts to remove asbestos from schools.[38] This has been in direct response to parental apprehension concerning the health of their children. There is substantial evidence that the possibility of developing mesothelioma is linked to the amount of time that has elapsed since first exposure, meaning that the younger a person's age when initially exposed to asbestos, the greater the lifetime risk of developing mesothelioma.[39] But acknowledging this fact, and even allowing for the hypothesis that the very young might be more susceptible to asbestos disease,[40] the health risk to children remains insignificant because the level of exposure in asbestos-containing schools has in general been so low. The exceptions are found in those instances where loose asbestos is being actively disturbed or is falling from ceilings. It follows that, with these exceptions, the programme for removing asbestos from all asbestos-containing schools was not justified by the health risk posed to students. In some cases, the asbestos control work may have been justified in order to protect building renovation, maintenance, or custodial workers from exposure to asbestos if their work would have disturbed asbestos insulation. But neither the scale nor the pace of the school programme was warranted by the risk posed by most asbestos-containing schools to occupants or workers. If anything, the scale and pace of the programme significantly *increased* the risk to some workers directly engaged in control projects. Crash programmes invariably mean that inexperienced contractors and personnel will enter the field. A number of asbestos-control projects in schools were conducted in a manner which, according to the evidence before us, may have generated significant risk for the workers involved. There is no question, however, that the crash nature of the asbestos control programme in schools was occasioned by a climate of public apprehension; the actions of school boards and of the Ministry of Education, paralleled in other jurisdictions, show them to be responsive to public demand.

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[34] For the analysis of the health risk posed by asbestos exposure to building occupants, see Chapter 9, Section E.

[35] For data on the exposure of building workers, see Chapter 9, Sections B and D; and Chapter 10, Section C.

[36] For an explanation of regulation by procedure in contrast to regulation by control limit, see Chapter 10, Section A.

[37] See Chapter 10, Section C.3. For an analysis which compares the cost of asbestos removal while a building is still in use to the cost of asbestos removal just prior to demolition, see Chapter 9, Section E.

[38] For a discussion of the asbestos control programme in Ontario schools, see Chapter 9, Section E.

E. Asbestos Elsewhere

We are all exposed to asbestos because it is in the water we drink and the air we breathe. Counts of up to 4 million fibres per litre of drinking water have been found in southern Ontario municipalities (e.g., Sarnia and Metropolitan Toronto) and of up to 22 million fibres per litre in northern Ontario municipalities (Thunder Bay). The fibres are almost invariably extremely short, that is, below 1 micron in length.[41] The small dimension of these fibres, coupled with our finding that asbestos disease is occasioned by inhalation rather than ingestion, leads us to conclude that asbestos in drinking water is not a health hazard.[42] The same conclusion applies to asbestos in food and drink,[43] and we indeed recommend that a ban which the

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[39] For a discussion of the hypothesis that mesothelioma is time-dependent, see Chapter 5, Section E.4.

[40] See Chapter 5, Sections E.4 and H.

[41] For a description of the levels and dimensions of asbestos fibres in drinking water, see Chapter 11, Sections B.1 and B.2.

[42] For our analysis of the health effects of ingesting asbestos fibres, see Chapter 5, Section I.

[43] For a discussion of asbestos in food and beverages, see Chapter 11, Section B.3; the topic of asbestos in drugs is discussed in Chapter 11, Section B.4.

Liquor Control Board of Ontario has imposed on the use of asbestos filters in the production of beer, wine, and liquor should be lifted. (Ontario drinkers who remain apprehensive may wish to note that this ban was never effectively enforced on imported alcoholic beverages.)

We review asbestos in the outdoor air and note the prevalence of very short fibres and of low counts (a maximum of 0.0084 f/cc of all lengths) even in the vicinity of a Toronto expressway ramp, where vehicle braking action might be expected to release fibres.[44] We conclude that an ambient air quality objective such as the 0.04 f/cc guideline of the Ministry of the Environment remains important, not so much because of any health hazards that might be posed at such levels of exposure, but so that any unusual level of contamination can be identified and its source sought out. As for the adequacy of the 0.04 f/cc guideline, we recommend that the Ministry of the Environment should maintain it until internationally accepted methods for outdoor air measurement are adopted and permit a discerning review of its numerical level.

With respect to asbestos-containing consumer products, we express substantial concern over the pattern of regulation.[45] This pattern has been, in a word, erratic. Thus, for example, Health and Welfare Canada recommended that the use of asbestos in hand-held hairdryers should be discontinued although tests showed fibre release equivalent to normal background levels in outdoor air. Meantime, free-form asbestos, which may occasion significant fibre release, is unregulated and currently available at hardware stores throughout the country. As in many other aspects of Canadian life, the federal and provincial levels of government share jurisdiction over the regulation of consumer products. We prescribe that the Government of Ontario should take steps, preferably through federal-provincial collaboration, to sort consumer products into three categories. The first would be those that can release significant levels of asbestos fibres in normal use (e.g., loose-fill asbestos insulation); the second, those that possess the potential of releasing fibres from cutting or sanding or as a result of degradation (e.g., asbestos-cement sheet or asbestos gloves); the third, those products in which asbestos is sealed off or encapsulated (e.g., most appliances which contain asbestos insulation). The sale of products in the first category should be prohibited (unless there is no adequate substitute or the product is exclusively for the use of a manufacturing enterprise governed by the Regulation Respecting Asbestos). Products in the second category should be labelled and instructions made available on their safe use. Products in the third category should not be subject to regulation.

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[44] For a description of asbestos levels in the outdoor air, and for an elaboration of the points summarized in the balance of this paragraph, see Chapter 11, Section C.

[45] For an elaboration of this and the points summarized in the balance of this paragraph, see Chapter 11, Section A.

Finally, we single out the disposal of asbestos wastes as a source of concern, not so much because of any resulting contamination of outdoor air but to protect waste disposal workers from exposures that could pose a significant risk to their health.[46] Until it issued a new regulation in March of 1983, the Ministry of the Environment failed, in our view, to impose adequate waste disposal procedures. We prescribe further improvements to the new regulation. We also recommend that steps be taken to identify disposal sites in which substantial quantities of asbestos have been deposited and to develop policies governing any redevelopment that might take place on such sites.

F. Compensating Victims: Asbestos and Its Implications

To broach the subject of compensating the victims of industrial disease is to enter the realm of the Workers' Compensation Board (WCB). At the same time that we were inquiring into this agency's performance in the sphere of asbestos-related disease, it was the subject of another official study being conducted at the behest of the Minister of Labour by Professor Paul C. Weiler. We have endorsed, in whole or in part, a number of recommendations formulated by Professor Weiler. Given our sharply focused terms of reference, we have also probed deeply and prescribed specifically with respect to asbestos-related compensation.

No issues before this Commission proved more sensitive than those posed by workers' compensation. The Ontario WCB is a paradox. From one perspective, it is, in the sphere of asbestos disease, one of the most progressive compensation agencies in the world. This perspective is valid: the Ontario Board was the first workers' compensation agency in North America to compensate lung cancer in asbestos workers who did not also have clinical asbestosis; it has been the leader in compensating laryngeal and gastrointestinal cancer among asbestos workers; it has pioneered in rehabilitation and in outreach measures designed to identify workers whose disease arose from occupational exposure to asbestos. From the other perspective, the Board appears arbitrary and capricious, lacking in top-down direction and procedural fairness. Regrettably, we find this latter perspective eminently valid.

There is an explanation for the paradox of these conflicting and valid perceptions. Under its Act, the Board has been vested with broad discretion which it has used for enlightened ends. However, this same discretion, because it is inadequately structured and lacks confinement, is the breeding ground of arbitrariness.[47] Part of the problem lies with the Act rather than the Board. We, therefore, enthusiastically endorse Professor Weiler's recommendations that envisage a Workers' Compensation Appeals Tribunal, backed by Medical Review Panels, and issuing written, reasoned decisions. Like Professor Weiler, we urge that the Act be amended so that the Corporate Board will have a majority of outside, part-time directors, differing from him only in prescribing a somewhat larger membership.[48]

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[46] For elaboration, see Chapter 11, Section D.

Although part of the Board's problem lies with the Act, part is self-inflicted. Specifically in the sphere of asbestos-related disease, the Board has failed to use a section of its Act which creates a statutory presumption in favour of claimants whose disease is directly associated with exposure to an industrial process.[49] Because we view mesothelioma and asbestosis as asbestos-specific diseases, we recommend that the link between these diseases and asbestos exposure be covered by statutory presumption, and that this presumption, now rebuttable, be made irrebuttable.[50]

While the Board deliberately chose, some thirty years ago, not to avail itself of the statutory presumption in its own Act, it nonetheless attempted to structure its discretion by developing industrial disease guidelines. Claimants who satisfied the terms of these guidelines would be automatically compensated, provided the facts of their cases were verified; claimants outside the guideline would be scrutinized on a case-by-case basis. We review the guideline-setting process, with specific reference to asbestos-related disease, and find that it has been informal, internal, unsystematic, and piecemeal and that it has done nothing to dispel perceptions of Board arbitrariness.[51] We recommend that the formulation of eligibility rules should henceforth involve an Advisory Council on Industrial Disease Policy (ACIDP), empowered to appoint expert panels to assist in the development of such rules. More generally, we prescribe that the terms of reference of the ACIDP, whose chairman should be a member of the Corporate Board, should embrace all matters of industrial disease policy and point out specifically the role this Council could play in reviewing rehabilitation programmes, monitoring and improving outreach measures, and considering appropriate methods of financing the cost of industrial disease compensation.[52]

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[47] This is a recurring theme throughout Chapters 12 and 13.

[48] For elaboration of our views on appeals and on the structure of the Corporate Board, see Chapter 12, Section C.1.

[49] For elaboration, see Chapter 12, Section B.2.

[50] For elaboration, see Chapter 12, Section C.2.

[51] Our grounds for this finding are set out in Chapter 12, Section B.3.

[52] The structure and role of the ACIDP are set out in Chapter 12, Section C.3; for yet further elaboration on its role see in Chapter 14, the concluding portions of each of Sections A, B, and C.

Among numerous recommendations addressing asbestos-related compensation matters, we highlight, for the purpose of this overview, those which focus upon the procedural and substantive issues posed by the compensation of asbestosis.[53] This irreversible and normally progressive condition, which can itself be fatal and in any event substantially reduces life expectancy, is a classic instance of an industrial disease whose victims are eligible for partial disability benefits. For decades, the Board has used a committee of experienced chest physicians, currently known as the Advisory Committee on Occupational Chest Diseases (ACOCD), to diagnose claimants and determine each patient's quantum (percentage) of medical impairment. The quantum of impairment is determined in accordance with the degree of physical impairment that medicine considers to be clinically measurable. We consider at length the fact that medicine has come to recognize the reality of psychological as well as physical impairment. We then review evidence, from both medical literature and sworn testimony, that the diagnosis of a condition such as asbestosis has adverse psychological consequences for the patient. We consider as well the fact that the courts, in calculating tort liability awards, have recognized psychological impairment notwithstanding the extreme difficulties that can be posed by its measurement. On the basis of all these considerations, we conclude that victims of asbestosis should be compensated for their combined physical and psychological impairment. By directive of the Corporate Board, the ACOCD should henceforth determine, by clinical diagnosis, the class of physical impairment to which each claimant should be assigned. Three classes, internationally recognized in the medical literature, should be used: Mild, Moderate, and Severe. The percentage of impairment recognized for compensation purposes should be stipulated by the Corporate Board's directive rather than determined by the ACOCD. This percentage should recognize the combined physical and psychological impairment of asbestosis victims in accordance with the class of physical impairment to which they have been assigned by the ACOCD and should be, where the ACOCD finds Mild impairment, 30%; where it finds Moderate impairment, 60%; and where it finds Severe impairment, 100%.[54] In addition to this substantive matter, we consider numerous procedural issues posed by the operation of the ACOCD and its relationship to the Board and make the prescriptions we deem appropriate.

More generally, we take cognizance of the fact that the Board, to its credit, has a benefit of doubt policy to guide its adjudication process, provide observations on the extent to which this policy has been neither uniformly followed nor uniformly communicated, and conclude that benefit of doubt should be elevated to the level of the *Workers' Compensation Act.*[55] We address as well problems that beset the quality of Board communications with claimants and prescribe accordingly.[56]

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[53] For elaboration of all the points summarized in this paragraph, see Chapter 13, Section B.

[54] This recommendation has implications for the eligibility of the survivors of deceased asbestotics for death benefits; the matter of eligibility rules in this domain is addressed in Chapter 13, Section C.

In 1975, the Board launched a pioneering programme to rehabilitate employees whose past exposure to asbestos was deemed as warranting removal from further exposure.[57] We assess the factors that account for why this programme has been plagued by controversy and find that its deficiencies were indeed genuine, but that they are also understandable. However, we are distressed to find that removal and rehabilitation provisions contained in the Regulation Respecting Asbestos invite a repetition of these deficiencies, or worse, and we develop recommendations based on the lessons taught by the Board's Special Rehabilitation Assistance Programme.

The domain of outreach involves measures that seek to identify individuals whose disease or death was occasioned by industrial exposure and is therefore compensable by law.[58] We consider the evidence that asbestos-related deaths may be going uncompensated in Ontario and in this light find outreach measures to be necessary and important. We then review the Board's outreach measures and while we find them commendable, we conclude that such measures in future should be undertaken as a joint responsibility of the Board and of the Ministry of Labour.

Finally, we assess the extent to which a workers' compensation system can play a role in the prevention of industrial disease.[59] We find that the objective of prevention is closely associated with other major goals, notably equity among employers and justice. We consider the provisions of the *Workers' Compensation Act* and its regulations which, together with Board practices, govern the financing of compensation costs and the assessment of penalties. We find that the asbestos experience identifies severe deficiencies, whether from the standpoint of prevention, equity, or justice, and prescribe accordingly.

G. Learning from the Asbestos Experience

This entire Report is an exercise in learning from the asbestos experience and discerning the lessons it teaches about health hazards and their regulation. We close the Report with some observations concerning what the asbestos experience teaches us about hazard identification and about the process which is currently in place for designating hazardous substances in Ontario workplaces.

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[55] For elaboration, see Chapter 13, Section D.

[56] See Chapter 13, Section E.

[57] For elaboration of the points summarized in this paragraph, see Chapter 14, Section A.

[58] For elaboration of the points summarized in this paragraph, see Chapter 14, Section B.

[59] For elaboration of the points summarized in this paragraph, see Chapter 14, Section C.

The asbestos story demonstrates that the process of hazard identification can unravel slowly and that regulatory responses can lag behind the knowledge that slowly accumulates while a mounting toll of disease and death is borne by workers who are thereby cast in the role of human guinea pigs.[60] The alternative to this unacceptable state of affairs lies in swifter identification through new pre-screening techniques and swifter regulatory response. Fortunately, society now recognizes that factors in the human environment contribute importantly to premature death, that medical treatment is limited in what it can do for many of these conditions, and that prevention is the best approach for the control of many diseases. Recent though it is, society's appreciation of these facts is spawning more effective approaches to hazard identification and more timely and effective regulation. If the asbestos experience in the workplace offers stark testimony to the shortcomings of earlier societal views that were indifferent to disease prevention, it also speaks for the importance of being discriminating in the apprehension of hazards and in the formulation of regulatory responses. By the 1970s, society was sensitized to the contribution of the environment to disease and to the importance of prevention. However, as the enormity of the health disaster that was befalling asbestos workers came to be appreciated by the public in the 1970s, regulations were imposed and protective actions were taken which did not recognize that a substance that kills in the workplace may pose insignificant risks in the indoor or outdoor environment. This speaks clearly for an enhanced hazard identification and risk assessment capacity in government and prompts us to urge the creation, preferably on a national basis, of a hazard identification and risk assessment agency with sophisticated scientific capacity and open means of communicating with elected representatives, the media, and regulatory officials. We also consider the need for more co-ordinated communication of information by hazard-regulating agencies and prescribe accordingly.

A royal commission should probably be the last body to comment on any governmental process from the standpoint of the amount of time it consumes. We nonetheless comment on the process whereby the Ministry of Labour designates hazardous substances for regulatory purposes.[61] In the asbestos case, this process consumed more than two years. The open and participatory aspects of the designation process, which account in large part for its measured pace, are in our judgement entirely praiseworthy. Nonetheless, in order to provide workers with some additional protection during the long process of designation, we believe that the Ministry of Labour should consider the advisability of a two-stage procedure whereby a preliminary regulation would be issued at the time notice of intent to designate is given and remain in place until the final regulation is promulgated.

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[60] For elaboration of the points summarized in this paragraph, see Chapter 15, Sections B and C.

[61] For elaboration of the points summarized in this paragraph, see Chapter 15, Section D.

Was this Royal Commission really necessary? We pose the question with which we opened this chapter because it is for readers of this Report to answer. A final observation is in order: this Royal Commission lasted almost four years and cost the Ontario taxpayers $1.7 million.

Chapitre 1 Rapport de la Commission royale d'enquête sur l'amiante : Vue générale

A. Encore une commission royale d'enquête

Cette Commission royale d'enquête était-elle vraiment nécessaire?

Qu'elles le méritent ou non, les commissions royales d'enquête au Canada sont réputées des moyens lents, compliqués et coûteux pour étudier des questions plus ou moins importantes placées sous le signe de la controverse publique. Les sujets ainsi étudiés représentent une gamme de questions presque infinie : ils ont parfois été aussi spécifiques que les affaires d'une seule personne et aussi vastes que l'avenir économique du pays tout entier. Pour notre part, nous avons été nommés par le Gouvernement de l'Ontario pour nous pencher sur des questions de santé et de sécurité dans la province en ce qui concerne l'utilisation d'une seule substance : l'amiante. Depuis des années déjà, avant même que la Commission ne fût créée en avril 1980, on connaissait les effets ravageurs de l'amiante sur la santé. On n'avait guère besoin de nous pour confirmer cet état de chose.

Cette dure réalité a, à juste titre, provoqué l'appréhension générale. L'amiante est présent partout : il est dans l'air que nous respirons, il est dans l'eau que nous buvons. On l'utilise depuis des générations pour sa remarquable propriété de résister au feu et aux acides. Le Canada en est le plus grand producteur des pays occidentaux. Partout dans le monde, l'amiante a semé la maladie et la mort chez littéralement des milliers de personnes qui ont été exposées à ses fibres microscopiques dans l'exercice de leur profession : mineurs, ouvriers de moulin et de fabrique, poseurs d'isolant et travailleurs exerçant d'autres métiers. Il s'avère que ces personnes ont réellement sacrifié leur vie pour avoir de quoi vivre, ainsi que le proclame un ouvrage bien connu à ce sujet, *Dying for a Living*.[1] Quelles sont les conséquences de perpétuer ou non l'utilisation de l'amiante dans l'industrie? Et d'un point de vue plus général, sait-on si cette substance mortelle pour les travailleurs de l'industrie menace les autres à leur insu dans les bureaux, les aéroports, les écoles?

Qu'elle soit nécessaire ou non, la Commission n'est pas passée inaperçue. Sa première audience publique, tenue en octobre 1980, a attiré quelque 400 personnes et ce nombre a presque été égalé lors de la deuxième séance publique six semaines plus tard. Ces séances avaient été annoncées par les avis publiés dans 81 quotidiens et hebdomadaires de tous les coins de l'Ontario. Il était mentionné dans les avis que l'on pouvait obtenir sur demande une brochure documentaire sur notre plan d'activités. Envoyée en outre sur notre initiative à des personnes susceptibles de s'intéresser à notre mandat, cette brochure contenait une carte-réponse permettant au destinataire de s'abonner aux communications postales de la Commission, ou de signifier son intention de présenter un mémoire oral ou écrit. Plus de 2 000 personnes et organisations se sont inscrites et se sont ainsi tenues au courant des dates de chaque audience et de nos travaux de recherche. Pendant la semaine du 16 au 20 février 1981, nous avons siégé matin, midi et soir pour entendre les exposés oraux à Toronto, dans une ambiance aussi libre de formalités que possible, compte tenu de la présence intermittente des aveuglants projecteurs de la télévision. Les exposés sont venus de personnes de tout état et de toute condition, de l'ouvrier gravement malade ou de la veuve solitaire jusqu'au chef de grande entreprise ou dirigeant d'organisation ouvrière ou gouvernementale. D'autres audiences de ce genre ont suivi, le 27 mars 1981 à Windsor et le 8 juin 1981 à Toronto. Mais les commissions royales d'enquête ne se contentent pas de tenir des audiences sans formalités, du moins pas en Ontario; cela explique sans doute pour une bonne part leur lenteur et leur lourdeur légendaires.

> Suivant l'article 5(1) de la loi sur les enquêtes publiques,
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> La commission doit accorder, au cours de l'enquête, à quiconque justifie l'intérêt qui le lie directement à l'objet de l'enquête, l'occasion de témoigner et d'appeler des témoins pour les interroger ou les contre-interroger lui-même ou par l'entremise de son avocat à l'égard de témoignages pertinents.[2]

En octobre 1980, nous avons annoncé que les personnes intéressées à s'inscrire comme parties en règle suivant les dispositions prévues à l'article

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[1] Lloyd Tataryn, *Dying for a Living* (Toronto, Deneau and Greenberg Publishers Ltd., 1979).

[2] Traduction non officielle; texte anglais dans les Lois refondues de l'Ontario de 1980, chap. 411.

5(1) devaient le faire avant le 31 janvier 1981 et nous avons procédé à l'inscription en règle des vingt parties mentionnées à l'appendice A, à la fin du présent rapport. Nous avons décidé en outre que l'une des parties, les Victimes de l'amiante de l'Ontario (*Asbestos Victims of Ontario*), devait recevoir une aide financière publique pour la défrayer de ses frais de participation. Les Victimes de l'amiante de l'Ontario regroupe d'anciens travailleurs de l'industrie de l'amiante, leurs conjoints ou leurs veuves.

Les mois qui suivirent, les représentants des parties en règle ont rencontré périodiquement notre avocat-conseil pour établir par consensus la liste des témoins que l'on inviterait à témoigner. Le taux de réponse à nos invitations s'est avéré à peine inférieur à 100 pour cent. Les noms des témoins figurent à l'appendice B. Ces 53 hommes et femmes sont venus non seulement de l'Ontario et d'autres endroits au Canada, mais aussi de la Suède, du Royaume-Uni et des États-Unis pour témoigner sous serment. La plupart ont été interrogés et contre-interrogés pendant une journée entière et quelquefois deux. Une moitié environ de nos témoins étaient des scientifiques, constituant un répertoire international d'éminents spécialistes des effets de l'amiante sur la santé. Pour recueillir les témoignages en bonne et due forme, il a fallu 53 jours d'audience répartis principalement sur les mois de l'été 1981 et 1982.

Pendant que nous menions nos audiences, nous avons cherché, soit personnellement, soit par l'entremise de notre personnel, à étendre davantage nos connaissances par des observations directes et des contacts sans caractère officiel. Cela comportait, à titre d'exemple, des visites à certaines mines d'amiante du Québec, à l'École de médecine du Mount Sinai à New York, à certains établissements manufacturiers en Ontario et à quelques projets où on éliminait l'amiante des bâtiments. Nous avons aussi créé un programme de recherche pour mieux comprendre les questions qui s'imposaient à nous. Ces recherches ont produit deux volumes de documentation générale et dix études spécialisées constituant chacune un volume en soi, dont on trouvera les titres à l'appendice F; nous avons diffusé ces ouvrages dès leur publication afin de les soumettre à examen et à critique. Ces études touchent à toute une gamme de sujets, partant des difficultés techniques posées par la mesure des niveaux de poussières d'amiante et aboutissant à l'indemnisation des travailleurs atteints de maladies associées à l'amiante en Ontario. Comme les études ont commencé de paraître en février 1982, l'information qu'elles révélaient et le contexte qu'elles fournissaient vinrent se greffer utilement à nos audiences subséquentes. Le dernier jour d'audience officielle fut le 24 août 1982. Après avoir donné le temps aux parties en règle d'examiner les témoignages, nous sommes retournés une dernière fois en salle d'audience, pour entendre leur exposé final, le 28 janvier 1983. À part cette occasion, nous nous sommes enfermés pour délibérer avec notre directeur de recherche, notre avocat-conseil et notre coordonnatrice administrative, dans l'intimité de nos 12 études de recherche et de documentation générale, de nos 88 mémoires écrits et de nos 8 738 pages de communications orales et témoignages faits sous serment.

Ce rapport est le fruit de nos délibérations. Sa longueur et son style — genre littéraire que nous qualifions de «commissionnesque» — garantissent que fort peu de gens le liront de la première à la dernière page. Dans ce chapitre, nous essayerons de distiller nos constatations pour en livrer le strict essentiel. Les lecteurs qui — sous l'empire de la colère ou du ravissement, de la stupéfaction ou de la curiosité, ou pour quelque autre motif — désirent des explications n'ont qu'à se reporter aux notes de renvoi pour savoir dans quels chapitres ou parties de chapitres se trouvent l'information de base et le raisonnement que nous avons suivis. Comme nous avons été normatifs ainsi qu'analytiques, nous livrerons l'essentiel de nos 117 recommandations officielles dans le corps de ce chapitre.

B. Effets de l'amiante sur la santé

Les résidents de l'Ontario ont plus de raisons que les autres de ressentir l'appréhension que l'amiante a suscitée à l'échelle internationale. L'Ontario est le théâtre de ce que nous décrivons dans ce rapport comme une catastrophe de premier ordre au plan mondial en ce qui concerne la santé des travailleurs : l'usine Johns-Manville à Scarborough, dans la Communauté urbaine de Toronto. De 1948 à 1980, cette usine a utilisé un mélange de deux types d'amiante, le chrysotile et la crocidolite, pour fabriquer des tuyaux d'amiante-ciment. À divers moments de son exploitation, l'usine a aussi utilisé du chrysotile seul pour fabriquer de la planche d'amiante-ciment; de même, elle a employé, pour faire des isolants, du chrysotile et une troisième espèce d'amiante, l'amosite.[3]

En 1983, le nombre de décès dûs à l'exposition aux fibres d'amiante dans cette usine s'élevait à 68, si l'on se fie au nombre de demandes d'indemnité accordées par la Commission des accidents du travail.[4] Cette seule usine, où le nombre d'emplois n'a jamais dépassé 714 par année, a déjà entraîné plus de décès imputables à des maladies professionnelles que l'ensemble de l'industrie minière en Ontario, qui emploie au-delà de 30 000 travailleurs par année, ne provoque d'accidents mortels pendant une période type de quatre ans.

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[3] Pour une description des différents types d'amiante, voir chapitre 2, section B.

[4] Pour un exposé complet de l'expérience de la maladie et de la mort dans cette usine, voir chapitre 3, section A. Pour un compte rendu du coût de l'indemnisation accordée aux victimes de cette usine, voir chapitre 14, section C. Un effort spécial de réadaptation déployé pour les travailleurs de cette usine par la Commission des accidents du travail est examiné au chapitre 14, section A.

Le bilan des décès pour cette usine que l'on a fermée en 1980 offre un témoignage sévère quant à la nature de la maladie à longue période de latence.[5] Le nombre a progressé lentement et inexorablement parce qu'une période de quelque 10 à 30 ans, ou plus longue, sépare le décès de sa cause, qui remonte au début de l'exposition à l'amiante. La catastrophe a donc pris de l'ampleur avec le temps; ainsi, par exemple, entre août 1981 et août 1982, année centrale dans l'existence de la Commission, 5 anciens employés sont morts d'un mésothéliome, forme rare de cancer spécifiquement associée à l'exposition aux poussières d'amiante. On risque peu de se tromper, et cela est tragique, si l'on affirme que les ouvriers ayant déjà travaillé dans cette usine continueront de succomber aux méfaits de l'amiante et que la catastrophe n'est pas consommée.

Les décès associés à l'amiante surviennent par mésothéliome. Les décès par cancer du poumon, cancer gastro-intestinal et cancer du larynx peuvent avoir diverses causes, mais entre autres, l'amiante. Les décès peuvent survenir par amiantose; comme son nom l'indique, c'est une maladie strictement causée par l'amiante. C'est une variété de fibrose pulmonaire qui peut être mortelle en soi, mais elle agit plus communément en exposant les malades à des causes mortelles apparentées et, surtout, elle engendre un état d'infirmité irréversible et généralement évolutif. Au moment où l'usine Johns-Manville à cessé ses opérations en 1980, 113 de ses employés exposés à l'amiante avaient obtenu une pension d'invalidité partielle de la Commission des accidents du travail étant donné les divers degrés d'infirmité dont ils étaient affligés suite à l'amiantose.

La catastrophe dans cette usine se range parmi les pires incidents que l'on ait signalé dans la littérature internationale consacrée aux épidémies associées à l'amiante. Elle inscrit le nom de Scarborough sur une liste infamante où l'on retrouve Charleston, en Caroline du Sud; Rochdale, en Angleterre; et une poignée d'autres localités.[6] De tous les cas de décès et d'infirmité qui sont dûs à l'amiante et qui ont bénéficié d'indemnités de la part de la Commission des accidents du travail de l'Ontario, la moitié est attribuable à l'usine de Scarborough. Le reste résulte d'une exposition due à des situations professionnelles et des applications industrielles très diverses, comme, par exemple, la fabrication de masques à gaz pendant la guerre, l'usinage de freins et la construction de bateaux. En Ontario, les employeurs dont certains employés ont eu une maladie ou sont morts à cause de l'amiante se répartissent sur toutes les régions; à l'exclusion de

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[5] Pour une description des maladies associées à l'amiante et du phénomène de latence prolongée, voir chapitre 2, section D. Pour un examen plus poussé de l'intervalle de temps entre l'exposition initiale à l'amiante et la manifestation clinique de la maladie, voir chapitre 5, section E.4.

[6] Pour une analyse comparative de la maladie et de la mortalité chez les travailleurs de l'amiante dans diverses usines dans le monde, voir chapitre 5, section B.

Johns-Manville, seulement deux d'entre eux ont donné lieu à plus de 5 prestations d'indemnités.[7]

Il y a de quoi s'inquiéter au sujet de l'amiante en Ontario. Mais au fond, «au fin fond», que pense la Commission de la nature des menaces que l'amiante fait peser sur la santé? Le «fin fond» de notre pensée peut tenir dans les énoncés suivants :

1. Les maladies associées à l'amiante présentent un cas classique d'un phénomène appelé la relation dose-réponse. Plus l'individu est exposé à l'amiante (la dose), plus il est probable qu'il soit atteint de maladie (la réponse). Inversement, moins l'individu est exposé à l'amiante, moins il est probable qu'il soit atteint de maladie.[8] Cependant, quand l'exposition se situe à de faibles concentrations, l'évaluation du risque de maladie s'embrouille dans l'incertitude. Cette incertitude naît de plusieurs facteurs, notamment la difficulté de réaliser les mesures, les lacunes de l'épidémiologie et le manque de données.[9] Dès lors, la prudence exige de présumer que toute exposition comporte un risque de maladie.

2. Presque tout le poids de la preuve scientifique penche du côté établissant que l'inhalation (la respiration) de fibres d'amiante aboutit à la maladie. Aucune preuve formelle n'existe à l'effet que la maladie peut résulter de l'ingestion (l'avalement) de fibres d'amiante et il existe seulement des hypothèses très spéculatives pour expliquer comment l'ingestion rendrait malade.[10]

3. Les fibres d'amiante qui présentent la plus forte probabilité de nocivité par inhalation sont longues et minces. La «longueur» et le «diamètre» sont évidemment d'ordre relatif : les fibres se mesurent en microns et le micron est égal à un millionième de mètre. Les fibres d'amiante sont dangereuses quand elles dépassent 5 microns de longueur, ou peut-être 8, et que leur diamètre est inférieur à 1,5 micron, ou peut-être à 0,25 micron.[11]

4. Il y a de solides témoignages voulant que les fibres de crocidolite et d'amosite seraient plus nocives que les fibres de chrysotile, surtout parce qu'elles ont tendance à correspondre en longueur et en diamètre à la

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[7] Pour un exposé du volume des prestations accordées pour l'indemnisation de maladies et de décès chez divers employeurs en Ontario, voir chapitre 3, section A.

[8] Pour un examen des relations dose-réponse en ce que concerne les maladies associées à l'amiante, voir chapitre 5, section E et chapitre 7, section C.

[9] Pour un examen de ces facteurs, voir chapitre 4, section D.

[10] Pour un exposé de la preuve biologique quant à l'ingestion des fibres d'amiante, voir chapitre 4, section B.2. Pour un examen de la preuve épidémiologique, voir chapitre 5, section I.

[11] Pour une analyse de l'importance des dimensions de la fibre, voir chapitre 4, section B et chapitre 5, section D.

catégorie la plus dangereuse, mais aussi parce qu'elles ont une très grande aptitude à flotter dans l'air et, de là, à être inhalées par respiration. Il est également possible que la crocidolite et l'amosite soient plus nocives à cause de leur composition chimique, mais cette possibilité relève du domaine de la conjecture.[12]

5. Toutes les espèces de fibres peuvent causer n'importe quelle maladie associée à l'amiante, mais le mésothéliome est le résultat le plus probable d'une exposition à la crocidolite, quoiqu'il soit souvent l'aboutissement d'une exposition à l'amosite; toutefois, il n'est que faiblement associé à une exposition au chrysotile.[13]

6. Quel que soit le type d'amiante, la probabilité d'en respirer les fibres de dimensions particulièrement dangereuses dépend beaucoup du procédé d'utilisation de l'amiante. Ainsi, par exemple, la fabrication de garnitures de frein, qui comporte des opérations de forage et de polissage, risque beaucoup moins de produire des fibres nuisibles que la fabrication textile, qui comporte des opérations de filage et de tissage.[14]

7. Les victimes des maladies spécifiques dues à l'amiante ont été presque invariablement exposées à l'amiante à cause de leur travail. Cependant, il est indéniable que le mésothéliome atteint des personnes qui ont partagé le logis d'un travailleur à l'époque où celui-ci était exposé aux poussières d'amiante. Il est peu plausible que des personnes aient contracté une maladie parce qu'elles étaient exposées au voisinage d'une installation industrielle utilisant de l'amiante.[15] Nous n'avons pas trouvé de preuve à l'effet que la maladie atteint des personnes qui respirent de l'amiante à l'extérieur, en plein air, ou l'inhale parce qu'elles occupent des bâtiments où se trouve de l'amiante.[16]

8. L'amiantose est une maladie attribuable uniquement à l'exposition professionnelle à l'amiante. Il est fort évident qu'à un très bas niveau d'exposition à l'amiante, la fibrose pulmonaire, si effectivement elle peut être déclenchée, ne se développera pas jusqu'à produire une amiantose qualifiée

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[12] Pour un examen de la preuve qui indique que la crocidolite et l'amosite ont tendance à être plus nocives que le chrysotile, voir chapitre 5, section C. Pour un exposé de l'importance des dimensions de la fibre et de l'importance possible de la nature chimique de la fibre, voir chapitre 5, section D.

[13] Pour un exposé et une analyse de la preuve concernant l'incidence du mésothéliome en rapport avec les différents types de fibre, voir chapitre 5, sections C.4, C.6 et D; et chapitre 7, sections C et F.

[14] Pour un compte rendu de l'état de santé des travailleurs employés dans divers procédés d'utilisation de l'amiante, voir chapitre 5, section B et chapitre 7, section C. Pour un examen des effets du procédé sur les dimensions de la fibre et des effets qui en découlent pour la santé, voir chapitre 5, section D et chapitre 7, sections C et F. Pour une description des différents procédés industriels, voir chapitre 6, section C.

[15] Pour plus d'explications sur ces points, voir chapitre 5, section E.

[16] Voir chapitre 11, section C et chapitre 9.

par diagnostic clinique ou une manifestation quelconque qui puisse même provoquer le moindre malaise.[17]

9. Il n'y a aucun lien entre l'usage du tabac et le mésothéliome. L'amiantose ne peut pas être causée par le tabac, mais il est possible que le tabagisme accélère le développement du fibrome. Enfin, bien que l'amiantose puisse causer à elle seule le cancer du poumon, il est nettement prouvé que l'amiante et le tabac se conjuguent avec une telle puissance synergique pour engendrer cette maladie que fumer et travailler dans l'amiante équivaut à jeter de l'huile sur le feu.[18]

Nous avons fait plusieurs autres constatations quant aux effets de l'amiante sur la santé, mais ce qui précède se ramène aux faits saillants. Qu'est-ce que ces constatations comportent? Nous répondrons à la question en examinant d'abord l'amiante dans l'industrie fixe, ensuite l'amiante dans les bâtiments et, enfin, l'amiante dans d'autres contextes.

C. L'amiante dans les lieux de travail fixes

Les lieux de travail fixes, en l'occurence les mines et les usines de fabrication, constituent une compartimentation des activités reliées à l'amiante dans un but de réglementation, et cela pour diverses raisons. Premièrement, ces activités sont facultatives; elles pourraient être interdites si l'on jugeait qu'elles comportent des risques inacceptables. Deuxièmement, ces activités, du fait qu'elles sont circonscrites dans des lieux de travail fixes, se prêtent à des techniques de travail et à des équipements de contrôle qui réduisent la concentration des fibres d'amiante dans le milieu auquel les travailleurs sont exposés. Troisièmement, encore une fois parce que le travail s'effectue dans un lieu fixe, il est possible de surveiller de façon systématique la concentration de fibres d'amiante dans l'air. La méthode de mesure usuelle comprend : (i) le prélèvement d'un échantillon d'air par ce que l'on désigne comme la méthode de la membrane filtrante; (ii) le dénombrement des fibres captées dans chaque échantillon d'air à l'aide d'un microscope optique; et (iii) l'expression du nombre de fibres décelées en terme de tant de fibres par centimètre cube d'air (f/cc).[19]

Suivant les dispositions prévues par la *Loi sur la santé et la sécurité au travail*, l'amiante est une substance désignée et assujettie au règlement concernant l'amiante. Ce règlement enjoint les exploitants d'installations fixes de prendre toutes les mesures et dispositions nécessaires pour garantir que l'exposition des travailleurs aux poussières d'amiante soit réduite au plus bas niveau pratique et il stipule les normes à ne pas dépasser. Ces normes se situent à 1 f/cc pour le chrysotile, à 0,5 f/cc pour l'amosite et à 0,2 f/cc pour la crocidolite. Le règlement s'étend à tous les types d'installations fixes dans l'industrie utilisatrice d'amiante et n'établit aucune distinction entre les activités industrielles.

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[17] Pour un examen de cette affirmation, voir chapitre 5, section E.3.

[18] Pour un examen de la relation entre le tabagisme, l'exposition à l'amiante et la maladie, voir chapitre 5, section F.

[19] Pour une description détaillée et une analyse de la méthode de la membrane filtrante, voir chapitre 7, section B.

L'extraction minière de l'amiante et son utilisation dans toutes sortes de procédés de fabrication font partie de l'histoire de l'Ontario. L'exploitation minière, qui comporte l'extraction de l'amiante chrysotile, est à présent une activité marginale quasi inexistante; à notre connaissance, l'industrie manufacturière se limite à l'heure actuelle à l'emploi de l'amiante chrysotile et l'utilise surtout pour fabriquer des produits de friction, notamment les freins d'automobile.[20]

À quel risque s'exposent les travailleurs dans ces différents contextes et, quant à cela, dans toute autre activité impliquant l'utilisation de l'amiante en milieu industriel fixe? De tous les agents carcinogènes d'utilisation industrielle dans le monde, l'amiante est de ceux qui ont fait l'objet d'études les plus poussées. Il en résulte une base de données qui permet d'estimer le risque de maladie selon le niveau d'exposition professionnelle. Nous nous sommes servi de modèles d'incidence pathologique et de données tirées de maintes études épidémiologiques concernant des travailleurs exposés à l'amiante pour faire une projection du risque éventuel de maladie que l'exposition à tout type de poussière d'amiante associée à différents procédés de fabrication peut entraîner à divers degrés de concentration.[21]

En nous basant sur cette analyse, nous concluons qu'il nous est impossible de fermer les yeux sur toute activité de fabrication qui exige l'emploi de l'amiante crocidolite ou amosite.[22] Il devrait être interdit indéfiniment d'utiliser ces deux types d'amiante en Ontario.[23] En ce qui concerne le chrysotile, il faut distinguer les procédés industriels qui utilisent ce type d'amiante. La norme que le règlement concernant l'amiante impose à l'heure actuelle pour tous les procédés comportant du chrysotile est de 1 f/cc. Si cette norme est rigoureusement observée de sorte que, au niveau d'une moyenne pondérée sur 8 heures, elle dépasse rarement ce plafond, nous estimons que la moyenne du niveau d'exposition chez les travailleurs n'est pas supérieure à 0,5 f/cc.[24] Notre base de données nous permet de calculer le risque de maladie chez les travailleurs employés dans les milieux industriels suivants : mines et moulins; fabrication générale, sauf des produits textiles et des ciments; fabrication textile; fabrication de produits du ciment. Dans le cas de l'extraction minière et du traitement au moulin du chrysotile et dans le cas de la fabrication générale utilisant le chrysotile, nous constatons que le risque de maladie associé à l'exposition au chrysotile dans une limite normative de 1 f/cc, dûment appliquée, donne par projection un taux de mortalité bien inférieur au taux de mortalité résultant des accidents de travail dans l'ensemble de l'industrie manufacturière en Ontario. Par conséquent, ce risque se situe tout à fait dans les limites acceptables par la société en fait de risques professionnels.[25]

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[20] Pour une description de l'exploitation minière de l'amiante en Ontario, voir chapitre 6, section F; l'activité manufacturière est décrite au chapitre 6, section B.

[21] Ces estimations sont détaillées au chapitre 7, section C. Une description technique du modèle d'évaluation du risque figure à l'appendice du chapitre 7.

[22] Pour les critères que nous avons élaborés en vue d'évaluer quand une substance doit être soumise à une norme, quand elle doit être interdite, et la relation entre une substance donnée et les substituts possibles, voir chapitre 7, section E. La faisabilité et le coût des méthodes de contrôle de l'amiante sont examinés au chapitre 6, section C. Pour un examen des substituts et de leurs effets sur la santé, voir chapitre 6, sections D et E.

[23] Voir chapitre 7, section F.3. L'interdiction que nous recommandons prend la forme d'une prohibition absolue par le ministère du Travail, laquelle pourrait être abolie seulement si les risques pour la santé étaient considérablement réduits. Dans le cas de l'amosite, il faudrait fixer l'exposition maximum à 0,1 f/cc, soit un niveau inaccessible à l'heure actuelle (voir recommandation 7.14); dans le cas de la crocidolite, l'exposition maximum, si même l'on peut y songer, devrait être fixée à 0,02 f/cc (voir recommandations 7.12 et 7.13).

La fabrication de textiles avec du chrysotile est un cas entièrement différent. En-deçà de la limite de 1 f/cc dûment respectée, le risque de mortalité associé à cette transformation de l'amiante est estimé supérieur à la mortalité due aux accidents dans l'industrie de fabrication et il approche ou dépasse la mortalité d'origine accidentelle dans tout le secteur minier en Ontario. Au regard des accidents de travail, cette activité est depuis longtemps parmi les plus dangereuses de toutes en Ontario. Pour que le secteur de la fabrication de produits textiles comportant du chrysotile puisse réduire le risque de décès dû à la maladie au niveau qui prévaut dans les autres secteurs d'utilisation du chrysotile, il faudrait fixer la norme à 0,04 f/cc, c'est-à-dire à un niveau beaucoup trop bas pour que les techniques de mesure actuelles puissent en assurer l'application. Nous constatons par conséquent que ce procédé d'utilisation du chrysotile ne peut pas être toléré en Ontario et qu'il doit être interdit aussi longtemps qu'il ne sera pas possible de convaincre le ministère du Travail que sa pratique ne comporterait pas de risques plus grands que ceux posés par les mines et la fabrication générale utilisant le chrysotile.

Le risque que nous associons à la manipulation du chrysotile dans les mines et la fabrication générale peut être moindre que le risque d'accident dans la fabrication, mais il n'en demeure pas moins important; il constitue en effet un risque de maladie chez le travailleur de l'amiante qui, comme tout employé d'usine, s'expose à des accidents de travail mortels. Nous considérons donc ce qui suit comme essentiel pour que l'on tolère l'utilisation du chrysotile à une limite normative de 1 f/cc : (i) Tout travailleur doit être assidûment informé du risque qu'il court, ainsi que le prévoit le règlement concernant l'amiante. (ii) Le règlement doit être rigoureusement appliqué afin que la norme de 1 f/cc garantisse en réalité que l'exposition moyenne réelle soit de 0,5 f/cc. (iii) Le règlement, la *Loi sur la santé et la sécurité au travail*, ainsi que les pratiques et l'organisation du ministère du Travail doivent être modifiés afin que le gouvernement, l'assemblée législative, le public et, par-dessus tout, les travailleurs concernés soient assurés que le règlement touchant l'amiante chrysotile est appliqué efficacement.[26]

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[24] Pour la base de calcul, ainsi qu'un examen approfondi des erreurs d'échantillonnage et de mesure, de la fréquence de l'échantillonnage et autres questions du même ordre, voir chapitre 7, section B.

[25] Pour un examen approfondi de ce point et des points résumés dans le reste de ce paragraphe et dans le paragraphe suivant, voir chapitre 7, section F.

À cet égard, nous avons fait des recommandations expresses qui vont de la mise au point de méthodes plus exactes pour l'échantillonnage de l'air jusqu'à la participation accrue du travailleur au processus d'application et de surveillance. Nous signalons que l'une de nos recommandations propose la création d'une unité de surveillance des substances désignées, relevant du ministère du Travail, afin de renforcer son rôle d'inspection dans les cas où le lieu de travail abrite des substances désignées. À part son directeur, qui devrait être formé aux techniques d'enquête, l'unité n'exigerait pas du ministère d'embaucher du personnel nouveau, puisqu'elle ferait plutôt appel à des équipes interdisciplinaires composées de professionnels qualifiés en science et technologie à l'emploi de la Direction de la santé au travail; le directeur de l'unité assemblerait l'équipe voulue pour visiter sans préavis les usines utilisant des substances désignées.[27]

D. L'amiante dans les bâtiments

En Ontario, comme ailleurs, l'amiante a été utilisé couramment jusqu'en 1973 comme isolant soufflable ignifuge et insonorisant, et comme revêtement calorifuge pour les tuyaux et les chaudières de chauffage.[28] L'amiante est donc présent dans un grand nombre de bâtiments à multiples étages, y compris les immeubles à bureaux, les écoles, les usines et les aérogares. Le type d'amiante qui s'y trouve généralement est non seulement du chrysotile, mais aussi de l'amosite.

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[26] Pour un examen approfondi du règlement concernant l'amiante, le système de responsabilité interne et l'application, voir chapitre 8.

[27] Voir chapitre 8, section E.1.

[28] Pour un examen de l'application et des caractéristiques de l'isolant soufflable et de l'isolant pour tuyaux et chaudières de chauffage, des bâtiments où on en trouve et des types d'amiante utilisés, voir chapitre 9, section B.

Naturellement, la présence d'amiante chrysotile et d'amiante amosite dans les bâtiments a suscité une vive inquiétude chez le public. Cette appréhension est-elle justifiée? À cette question, nous répondrons en deux temps. Premièrement, l'amiante peut en effet poser un danger sérieux pour les ouvriers qui effectuent des travaux tels que l'enlèvement de l'amiante et la démolition de bâtiments, ou qui exécutent toutes sortes de travaux d'entretien des bâtiments.[29] Deuxièmement, l'inhalation de fibres de chrysotile et d'amosite par les occupants d'immeubles ne pose presque jamais de problème de santé, sauf peut-être quand on remue l'amiante et qu'on s'expose alors à un niveau particulièrement élevé de poussières, surtout lors des opérations d'enlèvement de l'amiante.[30]

Passons tout de suite à un examen plus approfondi de notre deuxième observation. Elle suit pour l'essentiel la ligne de pensée tracée par le comité consultatif du Royaume-Uni sur l'amiante (*United Kingdom Advisory Committee on Asbestos*) en 1979. Malgré cette constatation faite par les Britanniques, il semble que l'inquiétude publique quant au risque que l'amiante fait courir aux occupants d'un bâtiment ne veuille pas s'apaiser. Le cas échéant, c'est un argument de poids pour que les organismes publics agencent et coordonnent mieux leurs communications. Nous remarquons en particulier que l'inquiétude du public est attribuable en grande partie aux organismes publics chargés de contrôler l'usage de l'amiante, car ces organismes négligent de tirer au clair le facteur primordial qui domine les mesures du niveau d'amiante dans l'air des bâtiments. L'Ontario offre l'exemple parfait d'un domaine de compétence où deux organismes différents ont agi sans aucun souci de cohésion dans leurs communications. Le ministère du Travail de l'Ontario a promulgué à grand renfort publicitaire les normes obligatoires dans les lieux de travail fixes. Nous l'avons vu, la norme pour le chrysotile est de 1 f/cc. Parallèlement, le ministère de l'Environnement de l'Ontario a pour norme 0,04 f/cc en ce qui concerne la présence d'amiante dans l'air ambiant (habituellement assimilé à l'air dans les bâtiments). À première vue, il semble que la norme du ministère de l'Environnement est 1/25 de celle fixée par le ministère du Travail. Mais cela n'est pas vrai. La norme de 1 f/cc du ministère du Travail s'applique à un contexte où le dénombrement des fibres contenues dans l'échantillon d'air est effectué à l'aide d'un microscope optique. Par contre, la faible concentration de fibres envisagée par le ministère de l'Environnement s'appuie sur un dénombrement des fibres à l'aide d'un microscope électronique.[31] Le microscope électronique détecte des quantités de fibres invisibles au microscope optique. Ainsi, le comptage électronique des fibres d'amiante présentes dans l'air des bâtiments aboutira à un nombre plus élevé, l'équivalent d'un ordre de magnitude ou environ 10 fois plus de fibres que par comptage optique.[32] Il s'ensuit que la norme de 0,04 f/cc du ministère de l'Environnement se rapproche beaucoup moins de 1/25 et beaucoup plus de 1/250 de la norme établie au ministère du Travail.

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[29] Pour un examen des expositions à l'amiante que les travailleurs s'occupant des bâtiments ont encourues ou peuvent encourir, voir chapitre 9, sections B et D.

[30] Pour une analyse des expositions à l'amiante chez les occupants d'immeubles, voir chapitre 9, section D.

[31] Pour un examen de la norme concernant l'air ambiant, voir chapitre 11, sections C.4 et C.5.

Nous constatons qu'en plein air les concentrations de fibres en suspension sont souvent inférieures à 0,001 f/cc selon des mesures effectuées par microscope électronique, et qu'elles n'atteignent presque jamais 0,01 f/cc.[33] Par comptage optique, ces concentrations se situeraient dans l'ordre de magnitude inférieur. Chose sans doute plus importante, nous concluons que l'air dans les bâtiments dotés d'une isolation thermique à l'amiante soufflé atteint habituellement une moyenne inférieure à ce qui équivaut à 0,001 f/cc par comptage optique et touche des sommets rarement supérieurs à 0,01 f/cc. Supposons que l'air d'un bâtiment est contaminé par le chrysotile et par les fibres plus nocives de l'amosite, et faisons le calcul estimatif du risque que courent les occupants quand ce mélange de fibres atteint 0,001 f/cc. Le risque de décès consécutif à une maladie due à l'amiante chez une personne qui aurait occupé l'immeuble pendant 10 ans est 50 fois moindre que le risque de décès accidentel que cette personne courrait en voyageant 10 milles par jour en auto pour se rendre à cet immeuble matin et soir pendant la même période. À ce niveau, nous considérons négligeable le risque auquel les occupants d'un immeuble sont exposés et nous trouvons par conséquent que l'amiante présent dans l'air d'un bâtiment ne menacera presque jamais la santé des occupants.[34]

On ne peut toutefois pas affirmer la même chose à l'égard des travailleurs employés à des travaux de démolition, d'enlèvement de l'amiante ou d'entretien de bâtiments. Ces travailleurs peuvent être exposés aux fibres d'amiante à des niveaux qui dépassent de beaucoup la norme appliquée dans les installations industrielles fixes.[35] Le travailleur est ici exposé à l'amiante amosite et à l'amiante chrysotile. L'exposition, en ce cas-ci, peut se produire par vagues brèves mais intenses, capables de vaincre les défenses normales du poumon humain. Quand il s'agit de démolition, d'enlèvement et de certains travaux de réparation, le lieu de travail n'est pas fixe et s'apparente plutôt à un chantier de construction typique. Il s'ensuit que la protection du travailleur peut mieux se réaliser en précisant les méthodes de travail qu'en contrôlant la qualité de l'air dans le but de satisfaire à des normes, et nous donnons quantité de conseils à ce sujet.[36] Pour la démolition des bâtiments, le risque potentiel d'exposition est d'une telle magnitude que nous prescrivons expressément d'enlever tout l'amiante friable des bâtiments avant de les démolir.[37]

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[32] Pour un examen des rapports entre le comptage optique des fibres et le comptage des fibres par microscope électronique, voir chapitre 7, section B et chapitre 11, section C.1.

[33] Pour un examen des données concernant les concentrations d'amiante à l'extérieur, voir chapitre 11, section C.2.

[34] Pour une analyse du risque pour la santé que l'exposition à l'amiante pose pour les occupants d'immeubles, voir chapitre 9, section E.

[35] Pour des données sur l'exposition à l'amiante chez les travailleurs s'occupant des bâtiments, voir chapitre 9, sections B et D et chapitre 10, section C.

Depuis 1979, de nombreux conseils scolaires en Ontario, bénéficiant d'une subvention de quelque 26 millions de dollars du gouvernement provincial, ont déployé énormément d'efforts pour éliminer l'amiante des écoles.[38] Cela répondait directement aux pressions des parents inquiets pour la santé de leurs enfants. Il y a de solides arguments à l'effet que la possibilité d'apparition d'un mésothéliome est en rapport avec la période de temps écoulée depuis l'exposition initiale, ce qui signifie que plus une personne est jeune lorsqu'elle est exposée à l'amiante pour la première fois, plus elle risque d'avoir un mésothéliome au cours de sa vie.[39] Alors même que ce fait est reconnu et que par surcroît nous tenons compte de l'hypothèse selon laquelle les enfants seraient plus vulnérables aux maladies dues à l'amiante,[40] le danger pour la santé des enfants demeure négligeable, étant donné que l'exposition à l'amiante dans les écoles se situe habituellement à un niveau extrêmement bas. Les exceptions se rencontrent quand des morceaux d'isolant d'amiante sont remués ou lorsque ceux-ci se détachent du plafond. Il en résulte que, sauf exceptions, l'élimination de l'amiante de toutes les écoles n'était pas justifiée au regard du risque pour la santé des élèves. Dans quelques cas, il fallait sans doute contrôler l'amiante afin de protéger les travailleurs contre l'exposition à l'amiante si leurs tâches de rénovation, d'entretien ou de ménage les obligeaient à remuer de l'isolant d'amiante. Mais le risque que la plupart des écoles contenant de l'amiante posaient aux occupants ou aux travailleurs ne justifiait nullement une initiative à pareille échelle et allure. Si résultat il y a, cette initiative *a accru* sensiblement, par son étendue et son allure, le danger pour les travailleurs exécutant directement des opérations de contrôle. Les opérations d'urgence sont invariablement synonymes de l'entrée dans un domaine spécialisé d'entrepreneurs et de travailleurs inexpérimentés. Plusieurs opérations visant à contrôler l'amiante dans les écoles ont été effectuées d'une manière qui, à en juger par les témoignages devant nous, peut avoir exposé les travailleurs concernés à un risque important. Cependant, il ne fait aucun doute que l'urgence de l'opération visant à contrôler l'amiante dans les écoles est née d'un climat d'inquiétude publique; ainsi les conseils scolaires et le ministère de l'Éducation, tout comme leurs homologues ailleurs, ont agi de toute évidence en réponse à la demande du public.

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[36] Pour une explication sur la réglementation visant les méthodes au lieu d'une réglementation établissant des normes, voir chapitre 10, section A.

[37] Voir chapitre 10, section C.3. Pour une analyse qui compare le coût de l'enlèvement de l'amiante pendant que le bâtiment est encore utilisé au coût de l'enlèvement de l'amiante juste avant la démolition, voir chapitre 9, section E.

[38] Pour un examen du programme de contrôle de l'amiante dans les écoles de l'Ontario, voir chapitre 9, section E.

[39] Pour un examen de l'hypothèse selon laquelle le mésothéliome est fonction du temps, voir chapitre 5, section E.4.

[40] Voir chapitre 5, sections E.4 et H.

E. L'amiante dans d'autres contextes

Nous sommes tous exposés à l'amiante parce qu'on en trouve dans l'eau que nous buvons et dans l'air que nous respirons. On a compté jusqu'à 4 millions de fibres par litre d'eau potable dans des municipalités du Sud de l'Ontario (p. ex., Sarnia et la Communauté urbaine de Toronto) et jusqu'à 22 millions de fibres par litre dans des municipalités du Nord de l'Ontario (Thunder Bay). Les fibres sont presque toujours extrêmement courtes, c'est-à-dire inférieures à 1 micron de longueur.[41] Étant donné que ces fibres sont de petites dimensions et comme nous avons constaté que l'amiante provoque la maladie par inhalation et non par ingestion, nous arrivons à la conclusion que la présence de l'amiante dans l'eau potable ne met pas la santé en danger.[42] La même conclusion vaut pour l'amiante dans les aliments et les boissons,[43] et nous recommandons en effet que la Régie des alcools de l'Ontario lève l'interdiction frappant l'utilisation de filtres d'amiante dans la fabrication de la bière, du vin et des spiritueux. (Les consommateurs d'alcool en Ontario qui restent inquiets à ce sujet pourront noter que cette interdiction n'a jamais été réellement appliquée pour les boissons alcooliques importées.)

Nous avons examiné la question de l'amiante en plein air et nous avons noté la prévalence de fibres très courtes et de quantités peu élevées (un maximum de 0,0084 f/cc de toutes longueurs), même dans le voisinage d'un rampe d'autoroute à Toronto, endroit où l'on pourrait s'attendre à ce que le freinage des véhicules libère des fibres.[44] Nous concluons qu'un objectif visant la qualité de l'air ambiant, telle la norme de 0,04 f/cc adoptée par le ministère de l'Environnement, demeure important, non pas tellement parce qu'il y aurait du danger pour la santé à des niveaux d'exposition semblables, mais plutôt parce qu'on peut alors signaler tout écart inhabituel du niveau de pollution et en chercher l'origine. Quant à savoir si la norme de 0,04 f/cc est adéquate, nous recommandons que le ministère de l'Environnement la maintienne jusqu'à ce que la communauté internationale reconnaisse des méthodes universelles pour mesurer l'air extérieur et pour permettre de discerner le sens des relevés numériques.

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[41] Pour une description des niveaux et des dimensions des fibres d'amiante dans l'eau potable, voir chapitre 11, sections B.1 et B.2.

[42] Pour une analyse des effets sur la santé dûs à l'ingestion de fibres d'amiante, voir chapitre 5, section I.

[43] Pour un examen de la présence de l'amiante dans les aliments et les boissons, voir chapitre 11, section B.3; la question de la présence de l'amiante dans les médicaments est examinée au chapitre 11, section B.4.

[44] Pour une description des niveaux d'amiante dans l'air extérieur, et pour plus d'explications sur les points résumés dans le reste de ce paragraphe, voir chapitre 11, section C.

En ce qui concerne les biens de consommation contenant de l'amiante, nous nous inquiétons passablement de l'état de la réglementation.[45] La réglementation est, en un mot, désordonnée. Ainsi, par exemple, le ministère de la Santé et du Bien-être social du Canada a préconisé de cesser d'utiliser l'amiante dans la fabrication des séchoirs à cheveux portatifs, bien que les tests indiquent un niveau de fibres libérées équivalent aux niveaux normalement présents dans l'air extérieur. Pendant ce temps, l'amiante à l'état libre, qui peut occasionner la libération d'importantes quantités de fibres, n'est soumis à aucun règlement et se vend à l'heure actuelle dans les quincailleries partout au pays. Comme dans tant d'autres domaines de la vie au Canada, les biens de consommation sont sujets à une réglementation relevant de deux paliers de compétence, le gouvernement fédéral et le gouvernement provincial. Nous préconisons que le Gouvernement de l'Ontario prennent des dispositions, de préférence par voie de collaboration fédérale-provinciale, pour classer les biens de consommation en trois catégories. La première viserait les biens qui, à l'usage normal, seraient susceptibles de libérer des niveaux importants de fibres d'amiante (p. ex., l'isolant composé d'amiante libre); la deuxième comprendrait les objets qui libèrent des fibres quand on les coupe ou les sable, ou lorsqu'ils se dégradent (p. ex., panneau d'amiante-ciment ou gants d'amiante); la troisième catégorie regrouperait les produits dans lesquels l'amiante est incorporé ou enveloppé hermétiquement (p. ex., la plupart des appareils ménagers isolés à l'amiante). La vente des objets de la première catégorie devrait être prohibée (à moins qu'il n'existe pas de produit de remplacement adéquat ou que le produit soit destiné exclusivement aux entreprises manufacturières assujetties au règlement concernant l'amiante). Les objets de la deuxième catégorie devraient porter une étiquette et être accompagnés d'instructions pour en faire un usage sans danger. Les produits de la troisième catégorie ne seraient assujettis à aucun règlement.

Enfin, nous rangeons au nombre de nos préoccupations l'élimination des déchets d'amiante, non pas tellement parce qu'il en résulterait une pollution atmosphérique, mais plutôt parce qu'il faut protéger les travailleurs des services d'élimination des déchets contre les situations qui menaceraient leur santé.[46] Avant l'adoption d'un nouveau règlement en mars 1983, le ministère de l'Environnement a négligé, à notre avis, d'imposer des mesures appropriées pour l'élimination des déchets. Nous recommandons d'améliorer encore le nouveau règlement. En outre, nous préconisons de prendre des dispositions pour identifier l'emplacement des dépotoirs où l'on a déposé des déchets d'amiante en quantité considérable et d'adopter des politiques régissant tout réaménagement que l'on pourrait projeter pour ces espaces.

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[45] Pour plus d'explications à ce sujet et au sujet des points résumés dans le reste de ce paragraphe, voir chapitre 11, section A.

[46] Pour plus d'explications, voir chapitre 11, section D.

F. L'indemnisation des victimes : l'amiante et ses répercussions

En abordant la question de l'indemnisation des victimes de maladies professionnelles, on pénètre sur le territoire de la Commission des accidents du travail (CAT). Pendant que nous enquêtions sur les réalisations de cet organisme en ce qui concerne les maladies associées à l'amiante, une autre étude officielle, commandée par le ministre du Travail et confiée au professeur Paul C. Weiler, s'intéressait au même organisme. Nous avons endossé, intégralement ou en partie, plusieurs recommandations formulées par le professeur Weiler. Comme notre mandat couvrait un champ bien restreint, nous avons exploré en profondeur et nous avons fait des recommandations spécifiques quant aux actes de compensation se rapportant à l'amiante.

Nulle autre question abordée devant notre Commission ne s'est avérée aussi délicate que celle de l'indemnisation des travailleurs. La CAT de l'Ontario est un paradoxe. Vue sous un certain angle, elle est l'un des organismes les plus progressistes du genre dans le monde, pour ce qui touche aux maladies dues à l'amiante. Ce point de vue est valable : la Commission des accidents du travail de l'Ontario a été la première en Amérique du Nord à accorder une indemnité pour le cancer du poumon chez les travailleurs de l'amiante qui ne présentaient pas également les signes cliniques de l'amiantose; la CAT est le chef de file en ce qui concerne l'indemnisation des travailleurs de l'amiante atteints de cancer du larynx et de cancer gastro-intestinal; elle a ouvert la voie dans le domaine de la réadaptation et des mesures d'extension visant à identifier les travailleurs qui auraient pu souffrir de maladies dues à l'exposition professionnelle à l'amiante. Sous l'autre angle, la CAT paraît arbitraire et capricieuse, dépourvue de direction du sommet à la base et sans équité dans sa procédure. Malheureusement, ce dernier point de vue nous semble éminemment valable.

Il y a une explication à ce paradoxe issu d'orientations incompatibles et pourtant valables. De par sa loi constitutive, la Commission des accidents du travail est dotée d'un pouvoir discrétionnaire étendu qu'elle a employé à des fins éclairées. Cependant, ce même pouvoir discrétionnaire est le berceau de l'arbitraire parce que la CAT n'a pas la structure appropriée et l'encadrement nécessaire.[47] Une partie du problème vient de la loi et non de l'organisme qui en dépend. Nous endossons donc avec ferveur les recommandations du professeur Weiler qui envisagent la création d'un Tribunal d'appel des décisions concernant les accidents du travail, lequel serait soutenu par des Comités de révision médicale et rendrait par écrit des décisions argumentées. Comme le professeur Weiler, nous conseillons instamment de modifier la loi de sorte que le Conseil de direction de la CAT puisse se composer en majorité de commissaires externes à temps partiel, mais à la différence du professeur Weiler, nous prescrivons d'augmenter un peu plus le nombre de membres.[48]

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[47] Ce thème revient constamment tout au long des chapitres 12 et 13.

Bien que le problème de la CAT soit imputable en partie à la loi, l'autre part est de sa propre fabrication. Dans le domaine précis des maladies associées à l'amiante, la CAT a négligé de recourir à un article de sa loi constitutive, lequel crée une présomption légale en faveur des réclamants dont la maladie est directement associée à l'exposition à un procédé industriel.[49] Étant donné que nous considérons le mésothéliome et l'amiantose comme des maladies spécifiques à l'amiante, nous recommandons que le lien entre ces maladies et l'exposition à l'amiante soit garanti par présomption légale et que cette présomption, à présent réfutable, soit rendue irréfutable.[50]

Alors que la Commission des accidents du travail choisissait délibérément, il y a une trentaine d'années, de ne pas user de la présomption légale inscrite dans sa loi constitutive, elle a néanmoins cherché à structurer son pouvoir discrétionnaire en élaborant des directives relatives aux maladies professionnelles. Les réclamants qui satisfaisaient aux conditions stipulées dans les directives obtenaient automatiquement une indemnité, pourvu que les faits exposés dans leur demande fussent véridiques; quant aux demandes qui ne cadraient pas avec les directives, elles étaient examinées minutieusement, cas par cas. Nous avons passé en revue le processus d'établissement des directives, par rapport aux maladies liées à l'amiante, et nous constatons qu'il a été informe, interne, incohérent et incomplet et qu'il n'a contribué en rien à chasser l'impression que la CAT agit de façon arbitraire.[51] Nous recommandons que les règles d'admissibilité soient dorénavant formulées avec le concours d'un nouveau Conseil consultatif pour la politique relative aux maladies professionnelles (CCPRMP), lequel serait autorisé à créer des comités de spécialistes chargés d'aider à élaborer lesdites règles. Sur un plan plus général, nous recommandons que le président du CCPRMP soit nommé un des commissaires au Conseil de direction de la CAT et que le CCPRMP ait un mandat englobant tous les aspects de la politique relative aux maladies professionnelles. Nous recommandons en outre que ce mandat précise exactement le rôle du CCPRMP quant à la révision des programmes de réadaptation, quant à la surveillance et à l'amélioration des mesures d'extension, et quant à la recherche de méthodes appropriées pour financer les indemnités de maladie professionnelle.[52]

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[48] Nos vues concernant les appels et la structure du Conseil de direction de la CAT sont élaborées au chapitre 12, section C.1.

[49] Pour plus d'explications, voir chapitre 12, section B.2.

[50] Pour plus d'explications, voir chapitre 12, section C.2.

[51] Nous étayons ces constatations au chapitre 12, section B.3.

Parmi les nombreuses recommandations touchant les questions d'indemnisation reliées à l'amiante, nous soulignons, aux fins de cet aperçu général, celles qui visent les problèmes de procédure et de fond soulevés par les prestations aux victimes de l'amiantose.[53] Cette maladie irréversible et habituellement progressive, qui peut être fatale en soi et à coup sûr réduit sensiblement l'espérance de vie, est un exemple classique de la maladie professionnelle dont les victimes ont droit à des prestations pour invalidité partielle. Depuis des dizaines d'années, la CAT a recours à un comité de médecins expérimentés en matière de maladies respiratoires, appelé à présent le Comité consultatif des pneumopathies professionnelles (CCPP), pour établir le diagnostic des réclamants et déterminer le quantum (pourcentage) de diminution de capacité de gain chez chaque patient. Le quantum de diminution capacitaire est établi d'après le degré de dégradation physique que la médecine considère cliniquement mesurable. Nous examinons dans le détail le fait que la médecine en est arrivée à reconnaître l'existence d'une diminution psychologique aussi bien que physique. Ensuite, nous passons en revue les évidences, du côté de la littérature médicale et du côté des témoignages faits sous serment, qui démontrent que le diagnostic d'une maladie comme l'amiantose entraîne des répercussions psychologiques néfastes pour le patient. Nous examinons aussi le fait que les tribunaux, dans leur calcul des dommages-intérêts adjugés en responsabilité civile, ont reconnu la dégradation psychologique malgré les difficultés extrêmes que peut causer son évaluation. Nous fondant sur toutes ces observations, nous concluons qu'il faut indemniser les victimes de l'amiantose en fonction de la diminution capacitaire globale, c'est-à-dire tant psychologique que physique. Sur directive émanant du Conseil de direction de la CAT, le CCPP devrait dorénavant classer chaque réclamant, à la lumière d'un diagnostic clinique, par catégorie de diminution capacitaire physique. Trois catégories, ainsi que la littérature médicale internationale le reconnaît, devraient être utilisées pour qualifier la diminution capacitaire : légère, moyenne, grave. Le Conseil de direction de la CAT, et non pas le CCPP, devrait stipuler dans une directive le pourcentage de diminution reconnu aux fins d'indemnisation. Ce pourcentage devrait reconnaître la diminution capacitaire physique et psychologique globale des victimes de l'amiantose conformément à la catégorie de diminution physique dans laquelle le CCPP les aura classées, et il sera de 30% dans les cas jugés *légers* par le CCPP, de 60% dans les cas jugés *moyens*, et de 100% dans les cas jugés *graves*.[54] Outre cette question de fond, nous nous penchons sur de nombreuses questions de procédure que posent le fonctionnement du CCPP et ses relations avec le Conseil de direction de la CAT, et nous faisons les recommandations que nous jugeons appropriées.

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[52] La structure et le rôle du CCPRMP sont exposés au chapitre 12, section C.3; son rôle est élaboré davantage au chapitre 14, voir les parties finales des sections A, B et C.

[53] Tous les points résumés dans ce paragraphe sont élaborés au chapitre 13, section B.

Sur un plan plus général, nous prenons note du fait que la Commission des accidents du travail a le mérite de se guider sur un principe du bénéfice du doute pour prendre ses décisions, de faire des observations quant à savoir dans quelle mesure ce principe n'a été ni appliqué uniformément ni communiqué uniformément, et de conclure que le principe du bénéfice du doute devrait être porté au niveau de la *Loi sur les accidents du travail*.[55] Nous nous attaquons également aux problèmes qui gênent la qualité des communications de la CAT avec les réclamants et faisons des recommandations en conséquence.[56]

En 1975, la Commission des accidents du travail a lancé un programme innovateur pour réadapter les employés dont l'exposition antérieure à l'amiante semblait justifier de les soustraire à une plus ample exposition.[57] Nous évaluons les facteurs qui expliquent pourquoi ce programme a été marqué par la controverse et nous constatons que ses lacunes étaient véritables, mais par ailleurs compréhensibles. Cependant, nous déplorons de constater que les dispositions que l'on retrouve dans le règlement concernant l'amiante à l'égard du retrait et de la réadaptation invitent à répéter les mêmes erreurs, ou à faire pis, et nous formulons des recommandations fondées sur les leçons que l'on a pu tirer du Programme d'aide spéciale à la réadaptation créé par la CAT.

L'effort d'extension est du domaine des mesures visant à identifier les personnes dont la maladie ou la mort est causée par une exposition professionnelle et fait donc l'objet d'une indemnisation en vertu de la loi.[58] Nous envisageons le fait que des décès reliés à l'exposition professionnelle à l'amiante peuvent se produire en Ontario sans faire l'objet d'une indemnisation, et à la lumière de ce fait, nous jugeons nécessaires et importantes les mesures d'extension. Nous examinons ensuite les mesures d'extension prises par la CAT et, bien que nous les trouvions louables, nous concluons que les mesures de ce genre devraient à l'avenir incomber conjointement à la CAT et au ministère du Travail.

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[54] Cette recommandation a des répercussions sur l'admissibilité à des indemnités de décès pour les survivants des malades qui souffraient de l'amiantose avant leur mort; la question des règles d'admissibilité en cette matière est examinée au chapitre 13, section C.

[55] Ces points sont élaborés au chapitre 13, section D.

[56] Voir chapitre 13, section E.

[57] Les points résumés dans ce paragraphe sont élaborés au chapitre 14, section A.

[58] Les points résumés dans ce paragraphe sont élaborés au chapitre 14, section B.

Enfin, nous étudions dans quelle mesure un régime d'indemnisation des accidents du travail peut jouer un rôle de prévention à l'égard des maladies professionnelles.[59] Nous constatons que la prévention est étroitement liée à d'autres buts dominants, notamment l'équité chez les employeurs et la justice. Nous examinons les dispositions de la *Loi sur les accidents du travail* et des règlements afférents qui, avec les procédés en pratique à la CAT, régissent le financement des coûts d'indemnisation et l'évaluation des pénalités. Nous constatons que l'expérience due à l'amiante indique des lacunes graves, que ce soit au plan de la prévention, de l'équité ou de la justice, et nous faisons des recommandations pertinentes.

G. L'amiante : une expérience riche en enseignements

Ce rapport tout entier est un exercice d'apprentissage dicté par l'expérience de l'amiante; il faut savoir tirer les leçons qu'il propose à l'égard de la salubrité et de la réglementation. Le rapport se termine par des observations sur ce que l'expérience de l'amiante nous apprend au sujet de l'identification des dangers et du dispositif en place à l'heure actuelle pour désigner les substances dangereuses employées dans les lieux de travail en Ontario.

L'histoire de l'amiante décrit comment le dispositif d'identification des dangers peut se détraquer lentement et comment les fonctions de réglementation peuvent se laisser distancer par l'accumulation progressive des connaissances, alors que la maladie et la mort fauchent un nombre croissant de travailleurs relégués au rang de cobayes.[60] Cet état de choses inacceptable exige une alternative, c'est-à-dire une identification beaucoup plus prompte par de nouvelles techniques de pré-sélection et une réponse de surveillance réglementaire plus prompte. Heureusement, la société reconnaît à présent que des facteurs nés du milieu human contribuent sensiblement à une mort prématurée, que le traitement médical a ses limites en ce qui concerne les possibilités de guérison dans beaucoup de ces cas, et que les mesures préventives constituent la meilleure approche pour enrayer beaucoup de maladies. Toute récente qu'elle soit, la reconnaissance de ces facteurs par la société a commencé à fructifier et on assiste à l'avènement de méthodes plus efficaces pour identifier les dangers et d'une réglementation plus opportune et plus efficace. Si l'expérience de l'amiante en milieu de travail offre un témoignage sévère quant aux défauts des vues sociales antérieures, marquées par l'indifférence envers la prévention de la maladie, cette expérience proclame également l'importance du discernement dont il faut faire preuve pour définir les dangers et formuler les solutions réglementaires. Avec les années 1970, la société s'est sensibilisée à l'influence de l'environnement sur la maladie et à l'importance de la prévention. Toutefois, tandis que le public des années 1970 découvrait l'énormité de la catastrophe qui ravageait la santé des travailleurs de l'amiante, on imposait des règlements et on prenait des mesures de protection sans s'apercevoir qu'une substance mortelle en milieu de travail peut poser des risques insignifiants dans un milieu ordinaire à l'intérieur ou à l'extérieur. Cela témoigne en faveur d'une meilleure capacité d'identification des dangers et d'évaluation des risques de la part des pouvoirs publics et nous incite à recommander instamment que soit créé, de préférence à l'échelle nationale, un organisme chargé d'identifier les dangers et d'évaluer les risques, lequel serait doté de moyens scientifiques perfectionnés et aurait toute liberté de communiquer avec les représentants élus, les médias d'information et les corps publics investis d'un pouvoir de réglementation. Nous envisageons aussi la nécessité de mieux coordonner la communication de l'information émanant des organismes chargés de réglementer les situations dangereuses, et nous faisons des recommandations en conséquence.

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[59] Les points résumés dans ce paragraphe sont élaborés au chapitre 14, section C.

[60] Les points résumés dans ce paragraphe sont élaborés au chapitre 15, sections B et C

Une commission royale d'enquête est sans doute le plus malvenu des organes publics à faire des observations concernant la quantité de temps qu'exige toute opération d'ordre public. Nous le faisons néanmoins à l'égard du processus par lequel le ministère du Travail désigne les substances dangereuses à des fins de réglementation.[61] Dans le cas de l'amiante, il a fallu plus de deux ans pour y arriver. La participation ouverte qui caractérise ce processus et explique en grande partie sa marche mesurée est tout à fait louable à notre avis. Pourtant, si l'on veut assurer aux travailleurs davantage de protection pendant ce long processus de désignation, nous croyons que le ministère du Travail doit envisager l'opportunité d'une démarche en deux étapes suivant laquelle on adopterait un règlement préliminaire lors de la publication de l'avis d'intention de désigner et on le garderait en vigueur jusqu'à la promulgation d'un règlement définitif.

Cette Commission royale d'enquête était-elle vraiment nécessaire? Nous revenons sur la question posée au début du chapitre parce qu'il appartient aux lecteurs de ce rapport d'y répondre. Une dernière observation s'impose : la présente Commission royale d'enquête a duré presque quatre ans et elle a coûté 1,7 million de dollars aux contribuables de l'Ontario.

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[61] Les points résumés dans ce paragraphe sont élaborés au chapitre 15, section D.

Text of Formal Recommendations

There follows, numbered sequentially by chapter, the text of the 117 formal recommendations made by this Commission.

From: Part III
Asbestos in Fixed Workplaces

Chapter 7 Control Limits for Fixed Place Industry

7.1 **Whenever control limits that cannot be enforced by present measurement techniques are adopted, the Ministry of Labour should emphasize that section 17 of the Regulation Respecting Asbestos allows the substitution of alternative measurement techniques if the Ministry is convinced that the substitute techniques satisfy the criteria of section 17 of the Regulation.**

7.2 **The Ministry of Labour mounting method should be amended to replace the dimethyl phthalate and dimethyl oxalate preparation step with the acetone-Triacetin preparation step.**

7.3 The Ministry of Labour should require that all slides and remaining filter segments that represent workplace exposures in Ontario, whether counted by the Ministry of Labour or by a private firm, be catalogued and stored in Ontario for a period of five years after the sample is taken.

7.4 The Ministry of Labour method should be modified to incorporate tests of microscope resolution.

7.5 The Ministry of Labour should specify the Walton-Beckett graticule exclusively in the Code for Measuring Airborne Asbestos Fibres.

7.6 The Ministry of Labour method should be modified to provide a more rigid system of quality assurance, including the use of blank filters and recounting by other operators.

7.7 The fibre counting criteria published in the Asbestos International Association's (AIA) reference method should be adopted by the Ontario Ministry of Labour as an interim measure since they represent a current level of international consensus. If and when agreement is reached on counting criteria to be adopted for the International Organization for Standardization/Draft International Standard (ISO/DIS) method, these should then be considered for adoption by Ontario.

7.8 The Ministry of Labour should identify a laboratory to take responsibility for standardizing methods of asbestos fibre counting for workplace samples in Ontario and to certify individuals whose performance is judged by this laboratory to be satisfactory. Every attempt should be made to include worker representatives from Ontario asbestos manufacturing plants among such certified operators where the volume of measurement warrants it.

7.9 The Ministry of Labour should undertake a study of the relationship between the number of air samples taken and the distribution of those air samples among workers, work stations, and periods of the day or week, in order to determine the minimum requirements for an air sampling programme that will provide reasonable confidence that the data collected adequately reflect the maximum worker exposures in a workplace, the average exposure of those workers, and those work stations where particularly intensive monitoring may be desirable. This study should incorporate such statistical analyses as are necessary for the task. Upon completing the study, the Ministry should amend the Regulation Respecting Asbestos to provide more specific direction regarding the sampling programme that will satisfy that Regulation.

7.10 **The Ministry of Labour should conduct research using a scanning electron microscope alongside an optical microscope to explore the size distribution of fibres found in various workplaces.**

7.11 **The Government of Ontario should adopt regulations that would prohibit the operation of asbestos textile spinning and weaving plants in Ontario without the approval of the Ministry of Labour. This approval should be granted only if the Ministry is satisfied that the health risks faced by workers in the plant are far lower than the unacceptable risks estimated in this Report.**

7.12 **The Government of Ontario should adopt regulations that would prohibit the use of crocidolite asbestos in Ontario workplaces without the approval of the Ministry of Labour. This approval should be granted only if the Ministry is satisfied that the health risks faced by workers exposed to this asbestos are far lower than the unacceptable risks estimated in this Report.**

7.13 **The Code for Respiratory Equipment for Asbestos on Construction Projects should provide for the use of respiratory protection that will ensure that the worker's exposure does not exceed 0.02 fibres per cubic centimetre where crocidolite asbestos is in the air.**

7.14 **The Government of Ontario should adopt a regulation which prohibits the use of amosite in Ontario manufacturing unless and until it is demonstrated to the satisfaction of the Ministry of Labour that there is a measurement technology that will satisfactorily detect low amosite concentrations so that a 0.1 fibre control limit can be enforced with a reasonable degree of confidence.**

7.15 **The Code for Respiratory Equipment for Asbestos on Construction Projects should provide for the use of respiratory protection that will ensure that the worker's exposure does not exceed 0.1 fibres per cubic centimetre where amosite asbestos is in the air.**

7.16 **The Ministry of Labour, in applying the control limits of the Regulation Respecting Asbestos to automotive brake repair operations, should accept the use of wet methods of brake drum cleaning as *prima facie* evidence that the Regulation has been complied with.**

7.17 **The Ministry of Labour, in applying the control limits of the Regulation Respecting Asbestos to exposures of workers engaged in grinding and bevelling of brake drums in automotive brake repair operations, should accept as evidence that the Regulation is complied with:**

(i) that the operator is wearing a respirator while grinding and while cleaning the debris afterwards with wet methods, in shops where this operation occurs rarely; and

(ii) that the machines are equipped with local exhaust ventilation, in shops where grinding of brake shoes occurs frequently.

7.18 The Regulation Respecting Asbestos should be amended to specify that sections 6 through 16 do not apply to workplaces where the only exposure to asbestos arises out of the repair and maintenance of automotive brakes, unless grinding of brake shoes is performed frequently.

7.19 The Ministry of Labour should develop a modest information programme to communicate with the vast number of auto mechanics in Ontario the appropriate work practices as described in Recommendations 7.16, 7.17, and 7.18. A more intensive programme of communication should be designed to reach those mechanics working in brake specialty shops where, in particular, a considerable volume of grinding of new brake shoes is performed. The Ministry should also communicate appropriate practices to the secondary and post-secondary institutions in Ontario where auto mechanics training is currently provided.

Chapter 8 Beyond Control Limits: Protecting Health in Fixed Place Industry

8.1 The Ministry of Labour should undertake to identify instances of the successful operation of joint health and safety committees and the means whereby the factors that account for this success can be disseminated through information and training programmes, drawing upon the review by the Advisory Council on Occupational Health and Occupational Safety and such other analyses as are necessary.

8.2 The *Workers' Compensation Act* should be amended to provide that where an employer establishes or contributes from his own resources to the financing of a joint labour-management programme for the purpose of education in accident or disease prevention, and the Board is satisfied that the programme is serving the same purpose as an Accident Prevention Association programme, the Board may reduce the assessment for which the employer is liable as a member of a class represented by an Association.

8.3 The *Occupational Health and Safety Act* should be amended to provide that, where stipulated by regulation, members of a joint health and safety committee may be entitled to paid time from work for the purpose of occupational health and safety training; the Regulation Respecting Asbestos should be amended to stipulate that members of joint health and safety committees shall be entitled to paid time from work for the purpose of occupational health training in programmes approved by the Minister.

8.4 The Regulation Respecting Asbestos should be amended so as to require that every asbestos worker shall be informed of:

(i) the existence of a dose-response relationship for asbestos-related disease, namely, the lower the exposure, the lower the risk of disease; and

(ii) the fact that the Regulation establishes control limits as maxima and imposes on the employer the duty to take all necessary measures and procedures to ensure that the time-weighted average exposure of a worker to airborne asbestos is reduced to the lowest practical level.

8.5 Section 12 of the Regulation Respecting Asbestos should be amended to require that each time monitoring of airborne asbestos takes place, an appropriate summary statistic of the resulting fibre concentrations be calculated for each area of the plant, where areas are defined as portions of the plant in which dust levels are reasonably similar. The results for each area to be posted under section 12 should include these summary statistics. In addition, a summary of the average dust level in each area over the past one or two years should be calculated and compared with the current dust levels and with a weighted average of recent dust levels. The results of the monitoring and of this analysis should be posted as specified in section 12(a), but for a period of at least two months, or until rendered obsolete by subsequent measurements, rather than for the fourteen-day period specified in the existing Regulation.

8.6 The Regulation Respecting Asbestos should be amended so as to require that in every plant with a control programme, there shall be an individual certified by the Occupational Health Hygiene Service who will be designated as responsible for designing representative air sampling procedures; for seeing that the Code for Measuring Airborne Asbestos Fibres is respected; and for ensuring that equipment is properly calibrated.

8.7 **The Regulation Respecting Asbestos should be amended to require that the worker representative appointed pursuant to section 8(8) of the *Occupational Health and Safety Act:***

- **(i) have the opportunity to observe, on his plant tour, the calibration of air monitoring equipment;**
- **(ii) be consulted on the adequacy of representative air sampling procedures; and**
- **(iii) be afforded preparation time, together with office and secretarial assistance, beyond that already recognized for the preparation of minutes of committee meetings.**

8.8 **The Regulation Respecting Asbestos should be amended so as to require that every employer with a control programme must select persons who have supervisory positions for training in the nature of the hazards posed by asbestos, and in identifying and dealing with the specific workplace situations that may be most hazardous. The number of persons trained should be sufficient to ensure that one such person will be on duty in the plant at all times when its operation or maintenance entails worker exposure to asbestos.**

8.9 **The Ministry of Labour should review the content of the basic occupational health training it offers to Industrial Health and Safety Branch inspectors with a view to its adequacy and to whether it might be integrated with available joint labour-management health training programmes, and the Ministry should seek as expeditiously as possible to enhance the proportion of its inspectorate that has received occupational health training. As an interim measure, the Ministry should make every effort to ensure that the inspectors assigned to plants where designated substances are present are individuals who have received basic occupational health training.**

8.10 **The Ministry of Labour should create a Designated Substances Enforcement Unit (DSEU), headed by an official with the rank of Branch Director. This official would have the authority to launch special, unannounced inspection visits of plants in which designated substances are in use and to commandeer a multidisciplinary team of appropriate Ministry experts assembled separately for each inspection visit. Each DSEU team should have all the powers of a Ministry of Labour inspector.**

8.11 **The Regulation Respecting Asbestos should be amended to clarify that the examining physician under sections 13 to 16 shall be the physician designated by the employer, but that any employee shall** have the right to be examined, at the expense of the employer, by a physician selected by the employee, provided this physician certifies to the employer that he has conducted the examination in accordance with the Code for Medical Surveillance of Asbestos Exposed Workers.

8.12 The Regulation Respecting Asbestos should be amended so as to substitute for the role of examining physicians in maintaining the records, referred to in section 15(1), an obligation on examining physicians to forward the records or copies thereof to the Chief Physician, Occupational Health Medical Service of the Ministry of Labour. The system of control pursuant to which the Ministry maintains these records should be that prescribed by the Ontario Commission of Inquiry into the Confidentiality of Health Information.

From: Part IV
Asbestos in Buildings

Chapter 9 Problems of Asbestos in Buildings

9.1 The Ministry of Labour should prohibit the spraying of friable material containing more than 1% asbestos on a dry-weight basis.

9.2 The Ministry of Labour should assess, sponsoring studies where appropriate, the health risks associated with the fibres used as substitutes for asbestos in sprayed insulation, with a view to determining what regulations, *if any*, might be appropriate for the use of these substitutes.

9.3 The Ministry of Labour should ban the application of friable materials containing more than 1% asbestos by weight as insulation for pipes and boilers.

9.4 The Ministry of Labour should identify the compounds used as substitutes for pipe and boiler insulation, with a view to assessing any health risks associated with these substitutes and to determining what regulations, *if any*, might be appropriate for the use of these substitutes.

9.5 Legislation should be developed providing that, if significant quantities of friable insulation have fallen onto building surfaces and are being disturbed, the building owner must have the material tested to

determine whether it contains asbestos. This requirement should apply only to insulation installed or replaced before 1974.

9.6 Legislation should be developed providing that if the test mandated by the circumstances stipulated in Recommendation 9.5 shows that friable asbestos is present, the building owner must enclose, encapsulate, or remove the asbestos-containing material, following the work practices referred to in Recommendation 10.17.

9.7 The Minister of Labour should communicate with the Minister of Health with a view to establishing responsibility with respect to Recommendations 9.5 and 9.6.

9.8 Owners of private homes should be exempt from the duties set out in Recommendations 9.5 and 9.6 above.

9.9 The Ministry of Labour should communicate with workers who replace and maintain domestic space-heating boilers to inform them of the risks of working with asbestos and of the appropriate safety precautions for such work.

9.10 The Ministry of Labour should prepare a brief flyer describing the possible locations of asbestos on pipes and boilers in the home, the proper method of sampling suspect material, and any appropriate precautions should asbestos be found to be present.

Chapter 10 Protecting Workers from Asbestos in Buildings

10.1 The Ministry of Labour should frame a regulation under the *Occupational Health and Safety Act* respecting asbestos on construction projects and in buildings which includes building custodial and maintenance work. The Ministry should amend the existing Regulation Respecting Asbestos so as to specify that its terms do not apply to work activities encompassed by the new regulation.

10.2 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should require as follows:

(i) Before the demolition or renovation of any building the characteristics of which raise the possibility that it might contain friable asbestos, the building owner must cause the building or relevant part of the building to be inspected to determine

whether friable asbestos-containing materials are present. If friable asbestos-containing materials are present, Recommendation 10.11 should be followed in the case of demolition, and Recommendations 10.14 and 10.17 should be followed in the case of renovation.

(ii) Before performing maintenance or custodial work which would disturb friable fibrous material in any building the characteristics of which raise the possibility that it might contain asbestos, the building owner must cause the material to be disturbed to be tested for asbestos content. If the material is found to contain asbestos, the employer must cause work practices to be adopted which ensure the health and safety of the maintenance or custodial workers, as outlined in Recommendations 10.6, 10.14, and 10.17. Alternatively, the maintenance or custodial work may proceed without the suspect material being tested, provided that the work practices followed would be safe in the event that the material did contain asbestos.

(iii) The requirements of (i) and (ii) above should apply only to buildings in which insulation was installed or replaced before 1974.

10.3 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should require that in any case where a building is required by law to be inspected or tested for asbestos, the building owner must cause the results of the inspection or testing to be recorded as follows:

(i) in a public place to be designated by the Ministry of Labour;

(ii) in a document maintained on the building premises, under the care of the building owner or his representative. This document should be required to be shown on request to any government inspector, any building occupant, or any worker who is in the building for the purpose of exercising his trade and who might be exposed to friable fibrous materials; and

(iii) by placing signs or labels, in areas where asbestos material has been discovered, that clearly indicate the presence of asbestos and warn that the material should not be disturbed without using appropriate precautions.

10.4 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should provide for standardized laboratory procedures to be followed for analysis of bulk material taken from buildings. These procedures should be

reviewed from time to time so as to keep up with developing technology.

10.5 The Ministry of Labour should take steps to institute a register of individuals certified as competent to inspect buildings for asbestos. Certificates of competence should be awarded by the Ministry of Labour upon an applicant's having satisfactorily completed an approved examination. Incompetence or dishonest practices should result in the certificate being revoked. Only individuals named in the register should be permitted to undertake inspections for asbestos in buildings which are required by law.

10.6 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should require that once friable asbestos-containing material is found in a building, the building owner must institute a programme of management and custodial control. The scope of the programme should depend on the level of hazard created by the asbestos-containing material and should provide for the institution of safe work practices, including worker training therein, for all regular building custodial and maintenance workers. The programme should continue until the asbestos-containing material is removed from the building.

10.7 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should require that a written description of any management and custodial control programme, referred to in Recommendation 10.6 above, be submitted to the Ministry of Labour for approval. The Ministry of Labour should be empowered to approve or vary any programme so submitted, in order to ensure adequate protection for building workers.

10.8 The regulation pursuant to the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should govern the operations of a contractor who contracts with a building owner to take responsibility for worker training and safe work practices under a management and custodial control programme, so that the contractor would have the same responsibility as the building owner for ensuring that the relevant terms of the management and custodial control programme are met.

10.9 The Ministry of Labour should take steps to require that every contract for renovation, maintenance, or custodial work specify all known locations of friable asbestos-containing materials in the building which might be disturbed. If, in the course of the work, friable asbestos-containing materials of which the contractor was not informed are discovered in locations which could create some risk to

workers, adequate precautions for worker protection should be taken, at the sole expense of the building owner.

10.10 **The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should provide that where removal, enclosure, or encapsulation of sprayed asbestos-containing insulation is contemplated, encapsulation should only be an option if that insulation is in good condition and is sufficiently thin that it can be thoroughly penetrated by the encapsulant, or if removal or enclosure are not practicable.**

10.11 **The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should require that the owner of a building or structure ensure that prior to the demolition or partial demolition of a building or structure, all friable asbestos-containing materials which might be disturbed are removed from the building or structure, following procedures for removal referred to in Recommendations 10.14 and 10.17.**

10.12 **The Ministry of Labour should take steps to require that every contract for demolition specify all known locations of friable asbestos-containing materials in the building or structure which might be disturbed by the demolition work.**

10.13 **The Ministry of Labour should take steps to provide that all contracts for the demolition of a building or structure in Ontario shall be deemed to include a clause which provides for supplemental payment to the demolition contractor, on a time and materials basis, for all reasonable expenses incurred in removing any friable asbestos-containing material which was not specified in the demolition contract.**

10.14 **The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should distinguish among three levels of precautions:**

"Precautions A":

The minimum precautions, or "Precautions A," should include the following:

(i) **Workers must wear respirators in the hazardous work area [section 6, Proposed Regulation Respecting Asbestos on Construction Projects (PRRACP)].**

(ii) **Power tools used in the hazardous work area on asbestos-containing material may be operated only in conjunction with**

high efficiency particulate air filtered vacuums (section 9, PRRACP).

(iii) Workers must be educated as to the risks they face when working with asbestos and trained in the appropriate procedures for minimizing those risks (similar to section 11, PRRACP).

"Precautions B":

The intermediate precautions, or "Precautions B," should include, in addition to the precautions set out above as "Precautions A," the following:

(i) The Ministry of Labour must be notified before the hazardous work begins (section 4, PRRACP).

(ii) The work area must be separated from the rest of the project by a barrier capable of limiting the spread of asbestos fibres outside the work area (to be added to PRRACP).

(iii) Protective clothing must be worn by all workers (section 7, PRRACP).

(iv) Washing facilities must be available to workers (section 7, PRRACP).

(v) The hazardous work area must be properly and regularly cleaned (section 8, PRRACP).

(vi) No loose asbestos is to remain on the floors (section 10, PRRACP).

(vii) Asbestos-containing materials must be wetted prior to disturbing them, where practicable (section 13, PRRACP).

(viii) Asbestos work records must be kept for all workers involved in the hazardous work (section 14, PRRACP).

(ix) All workers involved in the hazardous work are to be subject to medical surveillance (sections 15–18, PRRACP).

"Precautions C":

The maximum precautions, or "Precautions C," should include, in addition to the precautions set out above as "Precautions 'A' and 'B,' " the following:

(i) The work area must be enclosed and sealed (section 13, PRRACP).

(ii) Showers and change rooms must be available to all workers (section 13, PRRACP).

(iii) The work area must be cleaned prior to the commencement of the work and subjected to a thorough two-stage cleanup after the work is completed (section 13, PRRACP).

10.15 The Ministry of Labour should consult with industry and labour to develop numerical definitions of areas of insulation to be classified as "small," "medium," and "large" for the purpose of determining which level of precautions, whether "Precautions 'A,' 'B,' or 'C' " referred to in Recommendation 10.14 above, is to be applied to asbestos control work projects of varying types and sizes.

10.16 The Ministry of Labour should consult with industry and labour to develop definitions of major, intermediate, and minor renovation, maintenance, and custodial projects for the purpose of determining which level of precautions, whether "Precautions 'A,' 'B,' or 'C' " referred to in Recommendation 10.14 above, is to be applied in each instance.

10.17 The Ministry of Labour should consult with industry and labour to determine, for the purposes of the regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings, the appropriate precautions to be applied to large, medium, and small control jobs and to major, intermediate, and minor renovations and maintenance and custodial projects, distinguishing, where appropriate, among wet-sprayed insulation, dry-sprayed insulation, and pipe and boiler insulation.

10.18 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should require that types of work in a hazardous work area which are not otherwise dealt with in that regulation shall only be performed in accordance with "Precautions A," described in Recommendation 10.14.

10.19 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should provide that where a worker frequently performs work calling for "Precautions A," described in Recommendation 10.14, asbestos work records should be kept for that worker and that worker should be subject to medical surveillance, as if the work called for "Precautions B."

10.20 The Ministry of Labour should, in consultation with industry and labour, prepare an alternative set of precautions for asbestos removal work outdoors that would allow the substitution of local

exhaust ventilation for the use of an enclosure in situations where enclosure is impracticable. This alternative set of precautions should be included in the regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings.

10.21 The regulation under the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings should provide that work procedures may be used which vary from those prescribed if the protection afforded by the variant procedures is equal to or exceeds the protection afforded by the prescribed procedures, or if it is demonstrated to the Ministry of Labour that those variant work procedures would always result in average worker exposures which, in the case of chrysotile, are at or lower than one-half the control limit and, in the case of crocidolite or amosite, are at or lower than one-half the exposure values prescribed in Recommendations 7.13 and 7.15. However, the use of variant work procedures should not in any way detract from requirements that would otherwise apply to the particular work situations regarding worker training, Ministry of Labour notification, asbestos waste handling, cleaning of the hazardous work area, wetting of asbestos, asbestos work records, or medical surveillance.

10.22 The Ministry of Labour should conduct air monitoring of control work, renovation work, and of maintenance and custodial work, from time to time, to determine whether the safety precautions referred to in Recommendations 10.14, 10.17, and 10.20 above are sufficient to ensure worker safety or are unnecessarily strict.

10.23 The Ministry of Labour should explore methods of communicating with control, renovation, maintenance, and custodial workers to inform them of the risks they face when working in buildings containing friable asbestos and of the appropriate procedures for minimizing those risks.

10.24 The Ministry of Labour should take steps to institute a register of individuals certified as competent to supervise the enclosure, encapsulation, or removal of asbestos-containing sprayed insulation and pipe and boiler insulation. Certificates of competence should be awarded by the Ministry of Labour; incompetence should result in the certificate being revoked.

10.25 The Ministry of Labour should take steps to require that no asbestos enclosure, encapsulation, or removal may take place without being supervised by a registered work supervisor. The Ministry of Labour should determine what exceptions to this requirement should be made for small jobs.

From: Part V
Asbestos Elsewhere

Chapter 11 Asbestos in the Environment

11.1 The Government of Ontario should take steps, either through federal-provincial collaboration or by having recourse to provincial legislative jurisdiction, towards the regulation of asbestos-containing consumer and construction products in accordance with the following scheme:

Products containing asbestos should be classified into one of three categories:

Category One:

This category would embrace loose asbestos which, during normal use or handling, is likely to release asbestos fibres in significant concentrations. An example of this is loose-fill asbestos insulation.

Category Two:

This category would embrace products containing asbestos which possess the potential to release asbestos fibres in significant concentrations in the event of misuse, improper handling, manipulation such as cutting or sanding, or as a result of product degradation. Products in this category would include asbestos-cement sheet; and asbestos gloves, simmering pads, and ironing board replacement sheets.

Category Three:

This category would embrace products containing asbestos which is sealed off or encapsulated so that the release of asbestos fibres is unlikely under foreseeable use. Products in this category would include most appliances which contain asbestos insulation and molded plastic products containing asbestos filler.

11.2 The following regulatory actions should be taken with respect to Categories One and Two:

(i) The manufacture, sale, and use of products in Category One, other than essential products for which there is no adequate substitute, should be prohibited in Ontario, unless the product is exclusively for the use of a manufacturing enterprise governed by the existing Regulation Respecting Asbestos.

(ii) All products in Category One, the manufacture, sale, or use of which are not prohibited in Ontario, and all products in Category Two, should be labelled with the following warning:

> **"Caution — Contains Asbestos — Breathing Asbestos May Be Dangerous to Your Health — Consult Pamphlet Available from Vendor for Proper Use."**

(iii) All purchasers of construction materials labelled in accordance with (ii) above should be provided with a pamphlet outlining the nature of the hazard posed by the product and the appropriate precautions to be taken in connection with the product.

11.3 Products in Category Three should not be subject to regulation.

11.4 The Ministry of Consumer and Commercial Relations should take steps to repeal the Liquor Control Board of Ontario ban on the use of asbestos filters.

11.5 The Ministry of the Environment, in co-operation with the Ministry of Labour, should financially support the development of internationally accepted methods for ambient air quality measurement and adopt such measurement methods when they have been adopted by such international bodies as the International Organization for Standardization.

11.6 The Ministry of the Environment should revise the emission standard for asbestos to specify concentrations of fibres longer than 5 microns. This standard should be consistent with the existing ambient air quality criterion for asbestos.

11.7 The Ministry of the Environment should amend the 1983 General — Waste Management Regulation made under the *Environmental Protection Act* so as to provide that the sections regulating asbestos waste apply only to asbestos waste generated by asbestos removal operations and by asbestos product manufacturing.

11.8 The Ministry of the Environment should amend the 1983 General — Waste Management Regulation made under the *Environmental Protection Act* so as to require that the Ministry be notified, prior to the transporting of asbestos waste, of the quantity of the waste, the nature and destination of the waste, the type of packing involved, and any other pertinent details.

11.9 The Ministry of the Environment should amend the 1983 General — Waste Management Regulation made under the *Environmental Protection Act* so as to require that precise records be kept as to the

location of deposited asbestos waste. The content and storage of these records should be specified by the Ministry.

11.10 The Ministry of the Environment should determine which disposal site operators are prepared to accept asbestos waste. If this determination indicates that there are not adequate disposal sites for receiving asbestos waste in the province, the Ministry of the Environment should either establish special asbestos disposal sites or consider requiring certain disposal site operators to accept asbestos waste.

11.11 The Ministry of the Environment should develop policies regarding the redevelopment of waste sites known to contain substantial quantities of asbestos.

11.12 The Ministry of the Environment should, where reasonably practicable, endeavour to identify sites in which substantial quantities of asbestos have been deposited.

From: Part VI
Compensating Victims: Asbestos and Its Implications

Chapter 12 Determining Eligibility for Asbestos-Related Disease Compensation

12.1 In line with the legislative exposure draft in the *White Paper on the Workers' Compensation Act*, the Corporate Board of the Workers' Compensation Board should be composed of a majority of outside directors.

12.2 Contrary to the legislative exposure draft in the *White Paper on the Workers' Compensation Act*, the number of Corporate Board members should be fixed at a maximum of twelve.

12.3 The provisions of section 80 of the legislative exposure draft in the *White Paper on the Workers' Compensation Act* concerning access to medical reports and opinions should be enacted in substantially their present form.

12.4 The provisions of the legislative exposure draft in the *White Paper on the Workers' Compensation Act* concerning the structure and procedures of the "Workers' Compensation Appeals Tribunal" should be enacted in substantially their present form.

12.5 The provisions of section 75(3) of the legislative exposure draft in the *White Paper on the Workers' Compensation Act* concerning the requirement that Appeals Tribunal decisions, including findings and reasons, be made in writing should be enacted into law, together with the supporting provisions requiring communication of decisions to the claimant, employer, or party of record.

12.6 The provisions of the legislative exposure draft in the *White Paper on the Workers' Compensation Act* concerning the structure and procedures of Medical Review Panels should be enacted in substantially their present form.

12.7 The Workers' Compensation Board, at the earliest opportunity, should advise the Lieutenant Governor in Council to amend Schedule 3 of the *Workers' Compensation Act* by inserting in the first column (the disease column) the words: "Asbestosis and Mesothelioma"; and in the second column (the process column) the words: "Any process involving the use of asbestos."

12.8 The Workers' Compensation Board should review Schedule 3 of the *Workers' Compensation Act* to ensure that it incorporates those diseases whose associated industrial process is necessary and sufficient to cause the disease.

12.9 In line with the legislative exposure draft in the *White Paper on the Workers' Compensation Act*, the provisions of Schedule 3 should be applicable to workers who have engaged in a scheduled industrial process at any time in their employment history.

12.10 Section 122(9) of the *Workers' Compensation Act* should be amended so as to stipulate an irrebuttable presumption in favour of the claimant.

12.11 The *Workers' Compensation Act* should be amended to provide for the creation of an Advisory Council on Industrial Disease Policy responsible for advising the Corporate Board on eligibility rules regarding disease compensation and on other matters of industrial disease policy. The Council's advice should be developed either as a matter of its own initiative or in response to an explicit request from the Corporate Board. The Council should be appointed by the Lieutenant Governor in Council and be composed of a Chairman, who would be an *ex officio* Director of the Corporate Board, and

not more than eight members appointed for stated terms. The legislation should authorize the Council to appoint expert Panels from time to time and should stipulate that both the Council's advice and the Corporate Board's response be public documents.

12.12 The Advisory Council on Industrial Disease Policy should, as a matter of high priority, transform the current Board guidelines on asbestos-related cancers into eligibility rules in accordance with the process prescribed in this Report.

12.13 The *Workers' Compensation Act* should be amended so as to:

(i) entitle individuals who contract mesothelioma, and are family members of asbestos workers who were domiciled with these workers at the time such workers were occupationally exposed to asbestos, to the same compensation benefits as the Act accords to employees; and

(ii) vest in the Workers' Compensation Board a statutory right to recover the costs of benefits paid to a family member of an asbestos worker from the employer of that worker.

Chapter 13 Processing Asbestos-Related Claims: Procedural and Substantive Issues

13.1 The Workers' Compensation Board, by directive of the Corporate Board, should promulgate as a matter of Board policy a rule for determining the quantum of compensable physical and psychological impairment arising from asbestosis in claimants diagnosed by the Advisory Committee on Occupational Chest Diseases (ACOCD) as suffering from this disease. The rule should instruct the ACOCD to assign such claimants to one of three classes of physical impairment defined by competent medical authorities as corresponding to Mild, Moderate, and Severe respiratory impairment and direct that the percentages to be used by the Claims Adjudication Branch for calculating awards for physical and psychological impairment be:

(i) where the ACOCD finds Mild physical impairment, 30%;

(ii) where the ACOCD finds Moderate physical impairment, 60%; and

(iii) where the ACOCD finds Severe physical impairment, 100%.

13.2 In assessing the implications of Recommendation 13.1 for the compensation of other diseases, the Workers' Compensation Board should address those diseases whose associated industrial process is their necessary and sufficient cause and whose manifestation involves chronic, irreversible disorders that shorten the individual's life expectancy.

13.3 The statutory definition of impairment contained in the legislative exposure draft in the *White Paper on the Workers' Compensation Act* should be reviewed to ensure that the statutory language of any relevant amendment to the *Workers' Compensation Act* is couched in language that includes psychological impairment.

13.4 The Corporate Board should promulgate a rule applicable to the procedural adjudication of asbestosis claims which stipulates that a physician of the Medical Services Division is to refer such claims to the Advisory Committee on Occupational Chest Diseases unless, in the opinion of the physician subject to check by the Claims Review Branch, the claim is deemed frivolous.

13.5 The Corporate Board should increase the membership of the Advisory Committee on Occupational Chest Diseases (ACOCD) to not fewer than twelve physicians recommended by the Executive Director of the Board's Medical Services Division after consultation with appropriate university and professional bodies, and it should direct that the ACOCD be organized into not fewer than four subcommittees, each composed of three physicians including a subcommittee chairman designated by the Corporate Board.

13.6 The Corporate Board should issue the following procedural rules to conduct the operation of the Advisory Committee on Occupational Chest Diseases:

(i) Each subcommittee is to have the authority to diagnose claimants and assign claimants to classes of physical impairment.

(ii) The clinical examination of every claimant is to involve the full membership of the subcommittee to whom the claimant is assigned.

(iii) Every subcommittee report is to be signed by the full membership of the subcommittee, with any dissent recorded.

(iv) In the event of subcommittee dissent, the relevant claim is to be reviewed by the chairmen of the three remaining subcom-

mittees who shall then make the finding with respect to diagnosis and class of physical impairment.

(v) The entire membership of the Advisory Committee on Occupational Chest Diseases is to review the findings of its subcommittees on a periodic basis.

13.7 The Corporate Board should adopt a rule whereby physicians engaged in the work of the Occupational Health Branch of the Ministry of Labour are ineligible for membership on the Advisory Committee on Occupational Chest Diseases and should take measures to ensure that the secretariat of this Committee is supplied solely by the Workers' Compensation Board.

13.8 The Occupational Health Branch of the Ministry of Labour and the Advisory Committee on Occupational Chest Diseases should continue to share the same x-ray and pulmonary function testing facilities, which should be accredited by an expert team of radiologists and respirologists at least every five years.

13.9 The Workers' Compensation Board, by directive of the Corporate Board, should promulgate an eligibility rule whereby survivors of deceased individuals who were rated by the Board as suffering Mild or Moderate impairment from asbestosis shall be entitled to benefits if death resulted from asbestosis, mesothelioma, lung, laryngeal, or gastrointestinal cancer, or *cor pulmonale*.

13.10 The *Workers' Compensation Act* should be amended so as to extend to claimants benefit of doubt in a manner consistent with that found in the *Pension Act* of the Parliament of Canada.

13.11 The Board should adopt a standardized form of communication with claimants which ensures:

(i) that all claimants for whom it is pertinent shall know that they have an explicit right to appeal and are informed of the procedures for launching an appeal; and

(ii) that all claimants whose disease is the subject of an eligibility rule shall have a copy of the pertinent eligibility rule.

13.12 The Workers' Compensation Board should adopt a standardized practice whereby:

(i) recipients of partial disability/impairment awards are informed of the rules pursuant to which their survivors may be eligible for benefits in the event of death; and

(ii) in all instances where the Board has been unable to grant survivor benefits within one month of death, the claimant's survivors shall be notified that the matter is under adjudication, provided with the pertinent eligibility rules, and asked to bring forward any relevant information.

Chapter 14 Rehabilitation, Outreach, and Prevention

14.1 The sections of the Regulation Respecting Asbestos and the portions of its Code for Medical Surveillance which deal with the removal and rehabilitation of asbestos workers should be thoroughly revised so as to:

(i) remove from examining physicians any role in determining whether asbestos workers are fit, fit with limitations, or unfit to work in an asbestos exposure;

(ii) direct examining physicians to inform the worker and the Workers' Compensation Board of any finding that a worker may have or has a condition resulting from exposure to asbestos;

(iii) vest an unequivocal right to refuse removal in the individual worker; and

(iv) specify that removal entails moving the worker to a position where an asbestos control programme mandated by the Regulation does not apply.

14.2 The Workers' Compensation Board should:

(i) take steps to ensure that all examining physicians under the Regulation Respecting Asbestos are familiar with the terms of the Asbestos Fibre Dust Effect guideline, the manner in which it was developed, and its limitations;

(ii) automatically set up a claim for any worker who, according to the finding of an examining physician, may have or has a condition resulting from exposure to asbestos;

(iii) refer every such claim to the Advisory Committee on Occupational Chest Diseases (ACOCD);

(iv) offer the option of removal and rehabilitation to workers who are confirmed, either by the ACOCD or upon appeal, as exhibiting asbestosis or dust effects; and

(v) review, in not less than ten years, and with the assistance of the Advisory Council on Industrial Disease Policy, the continuing appropriateness of offering removal and rehabilitation to asbestos workers.

14.3 The Memorandum of Understanding which constitutes the formal description of the operating relationship between the Ministry of Labour and the Workers' Compensation Board should be amended to recognize a joint Ministry-Board responsibility for the development and implementation of outreach measures designed to promote the identification of potential claims for industrial disease compensation.

14.4 The Ministry of Labour and the Workers' Compensation Board should attempt systematically to locate the residential addresses of all individuals who, because of previous asbestos exposure, have been examined by the mobile x-ray units of the Chest Surveillance Programme so that these individuals may be sent a card which they would be invited to present to their physicians whenever they wished. The card would inform the physician that its bearer was once under surveillance as an asbestos worker, alert the physician to be sensitive to any symptoms that might be indicative of asbestos-related disease, and invite the physician to contact the Ministry or the Board for records of his patient or other pertinent assistance.

14.5 The Ministry of Labour and the Workers' Compensation Board should systematically seek the co-operation of organizations such as the Ontario Medical Association and the Ontario Chapter of the College of Family Physicians of Canada to encourage all practising physicians to take work histories of patients whenever possible symptoms of asbestos-related disease are suspected and to inform such patients on how to initiate compensation claims. The Ministry and the Board should encourage these organizations to spread an understanding of the state of occupational medicine among physicians by all appropriate means, including local district council meetings and professional publications.

14.6 The Ministry of Labour and the Workers' Compensation Board, with the co-operation of the Ontario Hospital Association, should communicate to the chief executive officers of Ontario hospitals the

importance of taking steps that will promote, as a matter of hospital policy, the soliciting of occupational histories from patients who are admitted to hospital with symptoms of disease that may be asbestos-related.

14.7 The mandate of the Advisory Council on Industrial Disease Policy should include the role of monitoring the outreach efforts of the Ministry of Labour and the Workers' Compensation Board in an ongoing manner, assessing the relative success of these efforts, and advising on appropriate means of enhancing outreach.

14.8 The Workers' Compensation Board should levy against Johns-Manville Canada the maximum penalty assessment that can be made under section 91(7) of the *Workers' Compensation Act* in respect of the operation of the asbestos-cement pipe plant in Scarborough which, in 1980, was a member of rate group 137.

14.9 The *Workers' Compensation Act* should be amended to give the Workers' Compensation Board a statutory right of action against any employer the value of whose disease claims exceeds his contributions to the accident fund when the Board has reason to believe that the employer withheld from his employees knowledge which he possessed about the hazardous nature of a substance to which he exposed them. The amount of recovery by the Board should be statutorily fixed as twice the value of the employer's disease claims occasioned by the substance in question, one-half to be paid into the accident fund, the other half to the claimants involved.

14.10 In approaching the matter of any revisions in the experience rating of industrial disease, the Workers' Compensation Board should consider the financing of asbestos-related claims, until at least the turn of the century, as a special case. The Board should be sensitive to the proposition that the principle of horizontal equity speaks for greater rather than less experience rating among asbestos employers, and at a minimum does not countenance less experience rating than applies at present.

14.11 The Workers' Compensation Board should be sensitive to the potential of exposure charges on a selective basis, but should take notice that asbestos is likely not appropriate for such charges. Instances where such charges might appropriately be developed should be identified with the co-operation of the Ministry of Labour and the assistance of the Advisory Council on Industrial Disease Policy.

From: Part VII
Learning from the Asbestos Experience

Chapter 15 Identifying and Designating Hazardous Substances

15.1 The Government of Ontario should take steps, using its established channels of federal-provincial communication, to promote the expeditious development of a national agency for hazard identification and risk assessment that would serve the needs of governments at all levels of jurisdiction. This agency might be a federal agency, an agency jointly created by the federal government and the provinces, or a university-based centre or free-standing, non-profit institution with a mandate and funding provided jointly by the federal and provincial governments.

15.2 The Ministries of Labour and of the Environment should, as soon as possible after the release of this Report, prepare and disseminate an information sheet which explains, in the simplest possible terms, the differences between the measurement techniques used to derive fibre counts in fixed workplaces and the measurement techniques used to derive fibre counts in building air, and of the consequent irrelevance of making direct comparisons between the numerical asbestos control limits of the Ministry of Labour and the numerical guidelines of the Ministry of the Environment.

15.3 The Minister of Labour should make a formal reference to the Advisory Council on Occupational Health and Occupational Safety concerning what measures, including statutory amendment if necessary, should be taken to permit the preliminary regulation of designated substances while the designated substance process is underway.

Part II

Health Effects of Asbestos

Chapter 2 Asbestos and Disease

A. Introduction

There have been few substances in our environment whose possible adverse health effects have received as much attention as asbestos. The medical and scientific literature on asbestos is voluminous and continues to expand;[1] the health hazards of asbestos have been the focus of widespread media coverage; professional and educational conferences have been organized solely to consider its effects; asbestos has been the subject matter of several commissions of inquiry in other jurisdictions;[2] various groups of asbestos workers continue to be subjected to epidemiological study for evidence of excess disease; claims for asbestos-caused injuries have produced numerous lawsuits, particularly in the United States,[3] and frequent resort to workers' compensation agencies in this province and elsewhere; and exposures to asbestos at least in occupational settings are controlled in most jurisdictions.[4] Yet despite the close scrutiny to which the health effects of asbestos have been subjected, there is still much uncertainty and controversy on many of the most important issues. This is because the extensive study of the health effects of asbestos has refined the issues of concern. Certainly by the time this Commission was appointed, one no longer needed to ask whether asbestos was a health hazard. By April 1980, this had long been an established fact. By the time we commenced our inquiry, the inhalation of asbestos by workers who were exposed to it in their jobs had already left a tragic legacy of serious disease and death in many parts of the world.

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[1] For a fairly complete bibliography, see George A. Peters and Barbara J. Peters, *Sourcebook on Asbestos Diseases: Medical, Legal, and Engineering Aspects* (New York: Garland STPM Press, 1980); and Irving J. Selikoff and Douglas H.K. Lee, *Asbestos and Disease* (New York: Academic Press, 1978).

[2] For example: U.K., Advisory Committee on Asbestos, *Asbestos — Volume 1: Final Report of the Advisory Committee* (Simpson Report), William J. Simpson, Chairman (London: Her Majesty's Stationery Office, 1979); R.L. Zielhuis, rapporteur, *Public Health Risks of Exposure to Asbestos*, Report of a Working Group of Experts, prepared for the Commission of the European Communities (Oxford: Pergamon Press, 1977); and Québec, Comité d'étude sur la salubrité dans l'industrie de l'amiante, *Rapport final* (Beaudry Report), René Beaudry, J.C.P., Président (Québec: l'éditeur officiel du Québec, 1976).

[3] It has been estimated that by the end of 1982 more than 20,000 persons in the United States had instituted claims for damages for asbestos-related injuries. Almost all have been third-party product liability claims. See *The New York Times*, 28 January 1983, and see generally, the *Asbestos Litigation Reporter* (Edgemont, Pennsylvania: Andrews Publications, Inc., 1979–); and *Borel v. Fibreboard Paper Products Corporation* 493 F.2d 1076 (1973), cert. denied 419 U.S. 869 (1974). Such suits are basically statute-barred in Ontario by virtue of s. 8(9) of the *Workers' Compensation Act*, R.S.O. 1980, c. 539. See also, Chapter 14, Section C.1 of this Report.

But this legacy was the result of very high exposure levels many years ago. What is the health risk to workers at current, much lower levels of exposure? Are some types of asbestos more hazardous than others? Do some types of individual processes utilizing asbestos carry with them a greater risk of disease than others? Are the dimensions of the fibres important determinants of disease, and do they vary among individual processes? Do short yet intense exposures to asbestos of the kind to which construction, demolition, and building maintenance workers are likely exposed cause a disproportionate health risk? What is the health risk to children exposed to asbestos in schools; and, more generally, what is the health risk to all those exposed to asbestos outside the workplace? These were among the most difficult and salient questions confronting us as we began our deliberations.

In this chapter and the three that follow it, we endeavour to respond to these questions by examining the health effects of asbestos in detail. Chapter 2 provides a framework for this examination. It is devoted in the main to what is already known about asbestos — a description of what it is and its various types; of the types of processes in which it has been used; and of the diseases we know it can cause. In Chapter 3 we focus on the health effects of asbestos in this province and provide a brief history of the manner in which asbestos has been regulated in Ontario workplaces. In Chapter 4 we examine more intensively the sources of our knowledge concerning the health effects of asbestos and the criteria by which the medical and scientific evidence ought to be evaluated. Finally, in Chapter 5 we consider what we discern to be the most critical questions concerning the health effects of asbestos, weigh and assess the relevant evidence on these questions, and offer our best judgement as to the answers. In doing so we have been greatly assisted not only by a review of the considerable volume of medical and scientific literature which has been brought to our attention, but also by the sworn testimony of many of the internationally acknowledged experts on asbestos-related diseases,[5] testimony which was subjected to thorough cross-examination by the parties-with-standing at formal hearings of this Commission.

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[4] See Chapter 7, Section D of this Report.

B. What is "Asbestos"?

B.1 General Characteristics

Asbestos is not one mineral but a generic term used to describe a family of naturally occurring fibrous hydrated silicates divided on the basis of mineralogical features into two groups: serpentines and amphiboles. The important property of asbestos as compared to non-asbestiform varieties of silicates is the presence of mineralogically long, thin fibres that can be easily separated.[6] According to some definitions, there are as many as thirty varieties of asbestos, but only six are of commercial importance.[7] These, together with their chemical composition, are depicted in Figure 2.1.[8] Chrysotile, which is by far the most abundant, is the only type that belongs to the serpentine group. Crocidolite and amosite, the two other most commonly used fibres, together with anthophyllite, tremolite, and actinolite belong to the amphibole group.[9]

The word "asbestos" is derived from the Greek word meaning "inextinguishable" and the origin of its name reflects one of its principal characteristics: fire resistance.[10] But asbestos has many other qualities which enhance its commercial utility, among them tensile strength, durability, flexibility, and resistance to heat, wear, and corrosion.

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[5] For a list of the witnesses who gave evidence before this Commission, see Appendix B to this Report.

[6] Eric J. Chatfield, "The Problems of Measurement of Asbestos," in Ontario, Royal Commission on Asbestos, *Proceedings of The Royal Commission on Asbestos, Second Public Meeting, Friday, December 12, 1980*, reported by Lydia Dotto (Toronto: Royal Commission on Asbestos, 1981), Appendix A, p. 1. The asbestoses all have non-fibrous polymorphs of similar composition. See also, Gyan S. Rajhans and John L. Sullivan, *Asbestos Sampling and Analysis* (Ann Arbor, Michigan: Ann Arbor Science Publishers, Inc., 1981), p. 5.

[7] See Malcolm Ross, "The 'Asbestos' Minerals: Definitions, Description, Modes of Formation, Physical and Chemical Properties, and Health Risk to the Mining Community," in *Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods*, Gaithersburg, Maryland: 18-20 July 1977, NBS Special Publication 506 (Washington, D.C.: U.S. National Bureau of Standards, November 1978), pp. 49-63.

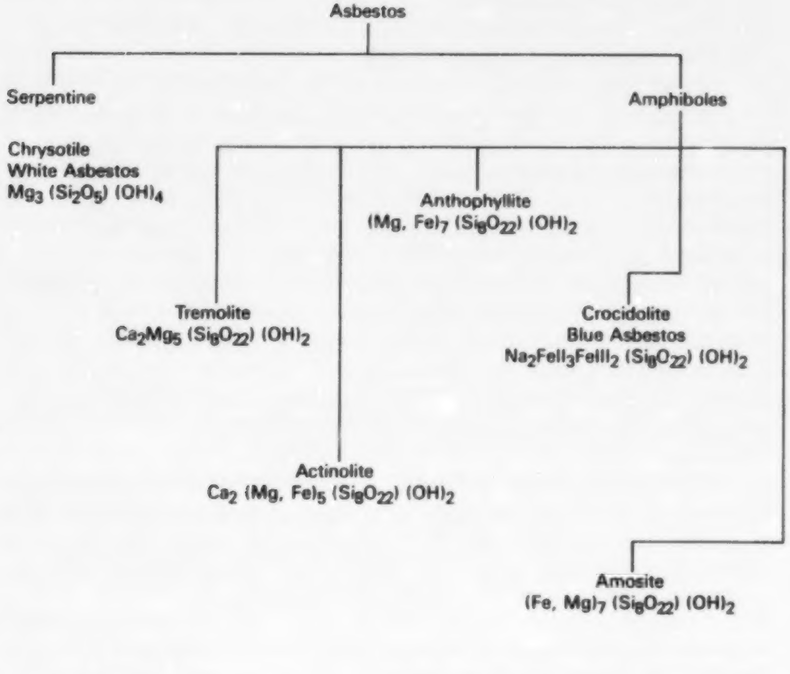
[8] Chatfield, "The Problems of Measurement of Asbestos," Appendix A, Figure 1, p. 2 and accompanying text, pp. 1-4.

[9] World Health Organization, International Agency for Research on Cancer Working Group, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, vol. 14: *Asbestos*, Lyon, France: 14-17 December 1976 (Lyon: IARC, 1977), pp. 11ff.

[10] Jack Zussman, "The Mineralogy of Asbestos," in *Asbestos: Properties, Applications, and Hazards*, vol. 1, eds. L. Michaels and S.S. Chissick (Chichester: John Wiley & Sons, 1979), chap. 2, p. 45.

Figure 2.1

Principal Varieties of Asbestos


Asbestos
Serpentine
Amphiboles
Chrysotile
White Asbestos
$Mg_3 (Si_2O_5) (OH)_4$
Anthophyllite
$(Mg, Fe)_7 (Si_8O_{22}) (OH)_2$
Tremolite
$Ca_2Mg_5 (Si_8O_{22}) (OH)_2$
Crocidolite
Blue Asbestos
$Na_2FeII_3FeIII_2 (Si_8O_{22}) (OH)_2$
Actinolite
$Ca_2 (Mg, Fe)_5 (Si_8O_{22}) (OH)_2$
Amosite
$(Fe, Mg)_7 (Si_8O_{22}) (OH)_2$

SOURCE: Dr. Eric J. Chatfield, "The Problems of Measurement of Asbestos," in Ontario, Royal Commission on Asbestos, *Proceedings of The Royal Commission on Asbestos, Second Public Meeting, Friday, December 12, 1980*, reported by Lydia Dotto (Toronto: Royal Commission on Asbestos, 1981), Appendix A, Figure 1, p. 2.

We are aware that the mineralogical classification of what is and what is not asbestos is complex, and as a result many different definitions of asbestos have appeared in the scientific literature. We are also aware that there are non-fibrous forms of minerals with essentially the same chemical composition as the asbestos varieties. In some cases the non-fibrous form has the same name as its fibrous counterpart, for example, tremolite.[11] The six varieties we have listed are deemed to be asbestos only when they have a fibrous form.[12] In addition to these six varieties, there are many minerals with fibrous crystal habits that occur naturally, which some authorities have included in the definition of asbestos. But these latter minerals either do not possess the useful characteristics generally attributed to asbestos or they do not occur in sufficient concentrations to be commercially exploited.[13] Having regard to our mandate "to investigate all matters relating to health and safety arising from the use of asbestos in Ontario," our use of the term "asbestos" will encompass those six asbestiform or fibrous minerals — chrysotile, crocidolite, amosite, anthophyllite, tremolite, and actinolite — which individuals are likely to come into contact with by reason of their commercial utility or natural occurrence.[14] Figure 2.2 shows the six types of asbestos pictorially. Table 2.1 sets out in tabular form the composition, properties, sources, and usage of these six fibre types.

B.2 Chrysotile

Chrysotile is a member of the serpentine group of minerals which also includes the minerals antigorite and lizardite. However, of these three serpentine minerals, only chrysotile occurs in an asbestiform or fibrous form that may be called asbestos. Chrysotile can also occur in a non-asbestos form that is chemically and structurally similar to the chrysotile asbestos form. The main difference between them is that the chrysotile asbestos form has crystallized as parallel fibres that can be easily separated into individual fibres or fibre bundles, whereas the non-asbestos form has

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[11] Ross, "The 'Asbestos' Minerals: Definitions, Description, Modes of Formation, Physical and Chemical Properties, and Health Risk to the Mining Community"; and Chatfield, "The Problems of Measurement of Asbestos," Appendix A, pp. 1–3.

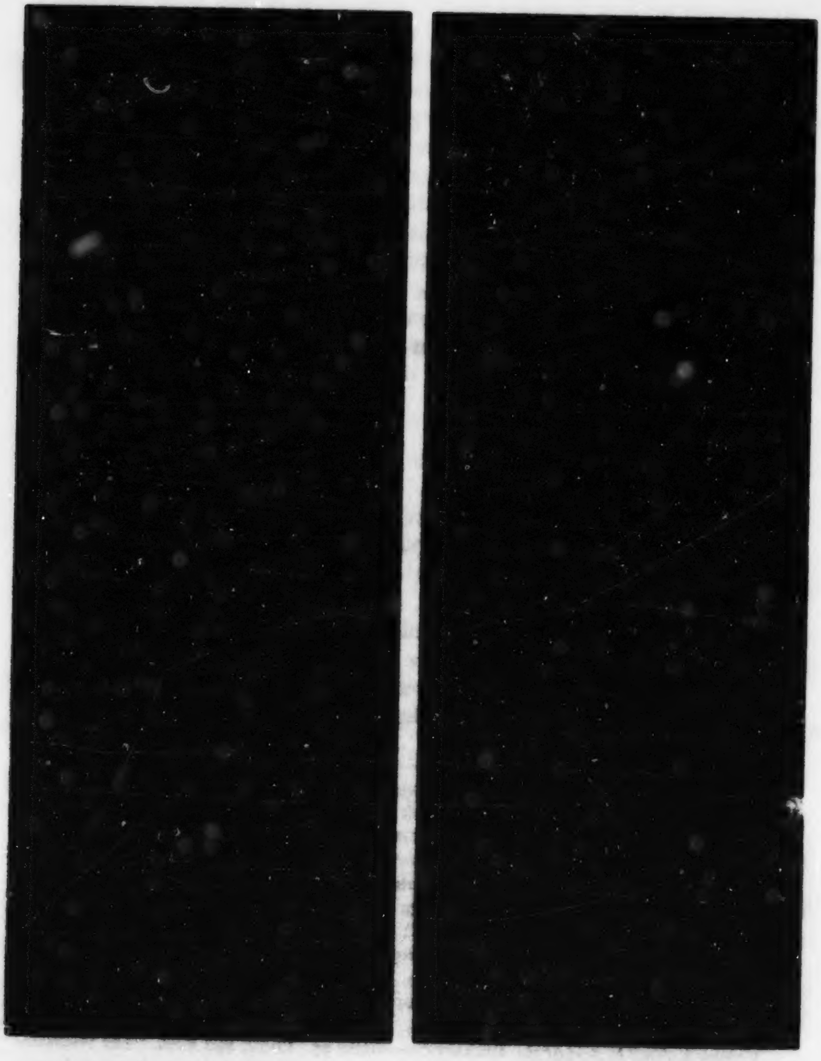
[12] The Ontario Regulation Respecting Asbestos defines "asbestos" as ". . . any of the following fibrous silicates: actinolite, amosite, anthophyllite, chrysotile, crocidolite or tremolite." See Regulation Respecting Asbestos, O. Reg. 570/82, s. 1(a), made under the *Occupational Health and Safety Act*, R.S.O. 1980, c. 321.

[13] World Health Organization, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, vol. 14: *Asbestos*, p. 11.

[14] The U.K. Advisory Committee on Asbestos took the same approach in its final report, as have the U.S. regulatory agencies. See U.K., Advisory Committee on Asbestos, *Asbestos — Volume 1: Final Report of the Advisory Committee*, pp. 11–12; and Ross, "The 'Asbestos' Minerals: Definitions, Description, Modes of Formation, Physical and Chemical Properties, and Health Risk to the Mining Community," pp. 49–53.

Figure 2.2

Types of Asbestos



asbestiform

non-asbestiform

SOURCE: R.T. Vanderbilt Company, Inc., Norwalk, Connecticut. Submitted for comment by the Royal Commission on Asbestos to Mr. Fred J. Wicks, Department of Mineralogy, Royal Ontario Museum, Toronto, Ontario, August 1983.

asbestiform

non-asbestiform

Table 2.1

Types of Asbestos: Composition, Properties, Sources, and Usage

Variety Property	Serpentine	Amphiboles				
	Chrysotile	Crocidolite (Riebeckite)	Amosite (Grunerite)	Anthophyllite	Tremolite	Actinolite
Essential composition	Hydrous silicate of magnesium	Silicate of Na and Fe with some water	Silicate of Fe and Mg; higher iron than anthophyllite	Mg silicate with iron	Ca and Mg silicate with water	Ca Mg Fe silicate; water
Crystal structure	Fibrous and asbestiform	Fibrous	Prismatic, lamellar to fibrous	Prismatic, lamellar to fibrous	Long and thin columnar to fibrous	Long and thin columnar to fibrous
Mineralogical structure	In veins of serpentine, etc.	Fibrous in iron formation	Lamellar, coarse to fine fibrous and asbestiform	Lamellar, fibrous asbestiform	Long, prismatic, and fibrous aggregates	Reticulated, long, prismatic crystals and fibres
Mineral association	In altered peridotite adjacent to serpentine and limestone near contact with basic igneous rocks	Iron-rich silicious argillite in quartzose	In crystalline schists, iron formation	In crystalline schists and gneisses	In Mg limestones as alteration product of magnesium rocks, metamorphic and igneous rocks	In limestone and crystalline schists
Colour	Usually white to green, grey, yellow, pink	Blue	Grey, yellow to dark brown	Yellowish-brown, greyish, white	Grey-white, greenish, yellowish, bluish	Greenish
Texture	Fine, soft to harsh, also silky	Soft to harsh, but flexible	Harsh and coarse but somewhat pliable	Harsh; soft to friable	Harsh or friable	Harsh, brittle

Table 2.1 (continued)

Types of Asbestos: Composition, Properties, Sources, and Usage

	Serpentine	Amphiboles				
Variety Property	**Chrysotile**	**Crocidolite (Riebeckite)**	**Amosite (Grunerite)**	**Anthophyllite**	**Tremolite**	**Actinolite**
Lustre	Silky	Silky to dull	Vitreous to pearly	Vitreous to pearly	Silky	Silky
Hardness	2.5-4.0	4	5.5-6.0	5.5-6.0	5.5	6+
Flexibility	High	Good	Good	Poor	Poor	Poor
Resistance to acids	Undergoes fairly rapid attack	Good	Attacked slowly	Very good	Very good	Attacked slowly
Resistance to alkalies	Very good	Good	Good	Very good	Good	Good
Spinnability	Very good	Fair	Fair	Poor	Poor	Poor
Tensile strength lb./in.^{2}	80,000- 100,000; 824,000 (max.)	100,000- 300,000; 876,000 (max.)	16,000- 90,000	4,000 and less	1,000- 8,000	1,000 and less
Filtration properties	Slow	Fast	Fast	Medium	Medium	Medium
Decomposition temperature (0°C)	450-700	400-600	600-800	600-850	950-1,040	620-960

Table 2.1 (continued)

Types of Asbestos: Composition, Properties, Sources, and Usage

Variety Property	Serpentine	Amphiboles				
	Chrysotile	**Crocidolite (Riebeckite)**	**Amosite (Grunerite)**	**Anthophyllite**	**Tremolite**	**Actinolite**
Major sources, present and past	Canada (Quebec, British Columbia, Yukon, Newfoundland, Ontario); U.S.S.R. (Urals, Siberia); Zimbabwe; Botswana; Swaziland; Australia (New South Wales); Cyprus; United States (Vermont, Arizona, California); China; Brazil	South Africa (North Western Cape, Transvaal); Bolivia; Western Australia	South Africa (Transvaal); India	Finland; United States (Georgia, Carolinas); South Africa; Bulgaria	Italy; South Africa; Pakistan; Korea	Taiwan; South Africa
World use, approximate %	90	6	3	<1	<1	—
Major industrial uses	Textiles, Cement products, Flooring, Friction materials, "Paper" products	Pressure pipes	Insulation products	None	None	None

SOURCES: Adapted from: Gyan S. Rajhans and John L. Sullivan, *Asbestos Sampling and Analysis* (Ann Arbor, Michigan: Ann Arbor Science Publishers, Inc., 1981), pp. 15–18; R.L. Zielhuis, rapporteur, *Public Health Risks of Exposure to Asbestos,* Report of a Working Group of Experts, prepared for the Commission of the European Communities (Oxford: Pergamon Press, 1977), p. 23; Margaret R. Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," *American Review of Respiratory Disease* 114:1 (July 1976): 187–227; and G. Oliver Vagt, *Asbestos* (Hull, Quebec: Supply and Services Canada, 1980).

crystallized as a complex tangle that cannot be separated into individual fibres or fibre bundles.[15]

Chrysotile differs from the amphiboles both structurally and in terms of chemistry. Chrysotile asbestos is composed generally of pliable, curly fibres made up of tiny individual fibrils which take the shape of a spirally wound tube.[16] It is the cylindrical structure of chrysotile that produces its fibrous character. Mineralogically, chrysotile is a layered silicate rolled up into a scroll and chrysotile asbestos is a number of these scrolls arranged parallel to each other. The curliness of the chrysotile fibre means that aerodynamically it behaves as if it were much thicker than its true diameter would suggest and hence neither easily becomes airborne nor efficiently penetrates through lung tissue in comparison to amphibole fibres. Chrysotile fibres have the ability to come apart very readily, splitting up into their tiny individual fibrils.[17]

The texture of chrysotile varies from soft, silky fibres to relatively harsh sticules. Although termed "white asbestos," chrysotile fibres may be green, grey, amber, pink, or white in colour. They are characterized by high tensile strength, a high resistance to alkalies, and, except for the harsh sticules, high flexibility and good spinnability.[18]

It is a common misconception that asbestos minerals are indestructible. In fact, all are susceptible to both chemical and thermal degradation. Chrysotile, when immersed in acidic fluid, loses its magnesium component, which can lead to the degradation of the fibres. Resistance to attack by other agents is in general excellent at temperatures below 100° centigrade, but this resistance deteriorates rapidly at higher temperatures. At temperatures of approximately 575° centigrade, the fibrous nature of chrysotile is destroyed, leaving inert particles.[19]

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[15] Letter from Mr. Fred J. Wicks, Department of Mineralogy, Royal Ontario Museum, Toronto, Ontario to the Royal Commission on Asbestos, 8 September 1983.

[16] See Peters and Peters, *Sourcebook on Asbestos Diseases: Medical, Legal, and Engineering Aspects*, p. A2; Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, p. 58; and RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 9.

[17] RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, pp. 52–54; RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, p. 96; Victor Timbrell, "The Inhalation of Fibrous Dusts," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 255–273; Victor Timbrell, "The Inhalation of Fibres," in *Pneumoconiosis: Proceedings of the International Conference, Johannesburg, 1969*, ed. H.A. Shapiro (Cape Town: Oxford University Press, 1970), pp. 3–9; and Paul Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," in *Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods*, p. 136.

[18] Zielhuis, *Public Health Risks of Exposure to Asbestos*, pp. 20–23; Eric J. Chatfield, "Measurement of Asbestos Fibres in the Workplace and in the General Environment," in Mineralogical Association of Canada, Université Laval, Québec: 20–22 May 1979, *Short Course in Mineralogical Techniques of Asbestos Determination*, ed. R.L. Ledoux (Toronto: Mineralogical Association of Canada, Royal Ontario Museum, May 1979), p. 116; and RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, pp. 61–62.

B.3 The Amphiboles

The amphibole group of minerals is composed of a large number of structurally similar but chemically different minerals, some of which occur in both fibrous and non-fibrous forms. One of the main features of the crystal structure of the amphiboles is their distinctive cleavage that produces elongated fragments when crystals are broken. Although the direction of fibre growth is parallel to the cleavage planes in amphiboles, the fibrous character is not produced by the cleavage. Amphibole asbestos fibres appear to be produced by specific geological environments that promote the development of fibres.[20]

Whereas chrysotile asbestos fibres are curly in shape, the amphibole asbestos fibres are straight and needle-like. Whereas chrysotile asbestos has a spirally wound, or concentric, layered silicate structure, the amphiboles have a chain-like silicate structure. Fibres of the amphibole series are generally more brittle and tend to cleave, that is, to split longitudinally much more readily than chrysotile. The amphiboles also appear to be more dusty than chrysotile, having a greater tendency to become airborne because of their straight, dart-like characteristics.[21] As a group, they are more resistant to acids than chrysotile and, save for crocidolite, more resistant to heat.

As we have already stated, there are five main types of amphibole asbestos — crocidolite, amosite, anthophyllite, tremolite, and actinolite — of which crocidolite and amosite have been commercially by far the most important.[22]

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[19] RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 55–56.

[20] Mr. Fred J. Wicks of the Department of Mineralogy, Royal Ontario Museum has suggested that there are more than 50 minerals that make up the amphibole group. Letter from Mr. Fred J. Wicks to the Royal Commission on Asbestos, 8 September 1983.

[21] RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, pp. 52–53; and Rajhans and Sullivan, *Asbestos Sampling and Analysis*, pp. 8–9.

[22] Mr. Fred J. Wicks of the Department of Mineralogy, Royal Ontario Museum has suggested that there is a sixth member of the amphibole group found in a fibrous form in a large enough amount to be mined as asbestos: namely, potassium winchite asbestos recently found in Texas. Letter from Mr. Fred J. Wicks to the Royal Commission on Asbestos, 8 September 1983.

Crocidolite, or "blue asbestos," is the fibrous variety of the mineral species riebeckite.[23] It has excellent acid-resistant properties and very high tensile strength, but only fair spinnability and poor resistance to heat.[24]

Amosite, which is not in fact a mineral name but an acronym for Asbestos Mines of South Africa, is mineralogically of the grunerite series.[25] Although known as "brown asbestos," it in fact varies in colour from yellow to grey to dark brown. It has a coarse texture, is highly resistant to heat, and is quite flexible, but may be susceptible to strong acids and alkalies. Its tensile strength is much less than that of chrysotile or crocidolite, and it has only fair spinnability.[26]

Anthophyllite fibres are yellowish-brown, greyish, or white in colour. They have a harsh texture, poor flexibility, poor spinnability, and low tensile strength, but a high resistance to acids and heat.

Tremolite varies in colour from grey-white to greenish-yellow or bluish. These fibres are quite resistant to acids and heat, but have poor flexibility and spinnability. Tremolite has a silky lustre and generally a harsh texture.[27]

Actinolite fibres have a greenish colour, a silky lustre, a harsh texture, and may be quite hard. However, they have a comparatively low tensile strength, poor flexibility, and poor spinnability.

The poor flexibility and spinnability of anthophyllite, tremolite, and actinolite are important reasons why there have been no major industrial uses of these fibre types.[28]

B.4 Commercial Deposits

Of the six fibre types, chrysotile dominates the world's commercial deposits of asbestos; 1980 production figures indicate that chrysotile alone accounted for approximately 90% of all asbestos produced in the world.[29] The major chrysotile deposits are found in the Soviet Union and in Canada, with the principal Canadian deposits being located in the Eastern Townships of Quebec and with significantly lesser deposits in Cassiar, British Columbia; Baie Verte, Newfoundland; Clinton Creek, Yukon; and in Northern Ontario. The deposits in Newfoundland and the Yukon are not being mined at the present time.[30] In the recent past there were four chrysotile mines operating in Northern Ontario, but only one is still in operation.[31] Munro Mines in Matheson, near Timmins, suspended operations in 1968, as did Reeves Mines in Reeves Township, also near Timmins, in 1975; and Matachewan Mines outside Kirkland Lake ceased production in 1977. Hedman Mines, a small operation with less than 20 employees located near Matheson, remains open but apparently mines asbestos only sporadically.[32]

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[23] Ross, "The 'Asbestos' Minerals: Definitions, Description, Modes of Formation, Physical and Chemical Properties, and Health Risk to the Mining Community," p. 50.

[24] See Table 2.1 in this chapter and the sources referred to therein.

[25] Chatfield, "The Problems of Measurement of Asbestos," Appendix A, p. 1.

[26] See Table 2.1 in this chapter and the sources referred to therein.

[27] Peters and Peters, *Sourcebook on Asbestos Diseases: Medical, Legal, and Engineering Aspects*, pp. A2–A4; Margaret R. Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," *American Review of Respiratory Disease* 114:1 (July 1976): 191; and Rajhans and Sullivan, *Asbestos Sampling and Analysis*, pp. 15–18. See also, Table 2.1 in this chapter.

[28] It is also true that there are few commercially exploitable deposits of these fibre types. See, for example, Zielhuis, *Public Health Risks of Exposure to Asbestos*, p. 18.

In addition to the Soviet Union and Canada, chrysotile is or has been mined in South Africa, Zimbabwe, Cyprus, Italy, the United States, China, Swaziland, and Brazil.[33]

The commercial production of amphibole asbestos is now almost completely confined to South Africa. Crocidolite is mined in the Cape and Transvaal regions of South Africa and to a very small extent in Bolivia. Australia has discontinued production of its crocidolite deposits, and a further deposit in Lusaka in Zambia has not been mined. Crocidolite, as of 1980, accounted for approximately 6% of the world's production of asbestos. Amosite is mined principally in the Transvaal and to a lesser extent in India and, as of 1980, accounted for approximately 3% of world production.[34]

Small amounts of anthophyllite were mined in Finland up to 1975 and are currently mined in South Africa, Bulgaria, and the United States. Fibrous actinolite is rare, but there is a small production from Taiwan and South Africa. Limited amounts of tremolite are mined in South Africa, Italy, Pakistan, and Korea.[35]

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[29] A.A. Hodgson, "Chemistry and Physics of Asbestos," in *Asbestos: Properties, Applications, and Hazards*, chap. 3, pp. 68–73; and G. Oliver Vagt, *Asbestos* (Hull, Quebec: Supply and Services Canada, 1980), p. 7.

[30] Roskill Information Services Ltd., *The Economics of Asbestos*, 4th ed. (London, England: Roskill Information Services Ltd., 1983), pp. 8–9, 21–23. The mine at Clinton Creek was closed in 1978 and the Advocate Mines in Baie Verte were closed in 1982.

[31] For a more detailed description of mining and milling in Ontario, see Chapter 6, Section F of this Report.

[32] Ontario Federation of Labour, Written submission to the Royal Commission on Asbestos, #35, January 1981, p. 50. See also, Chapter 6, Section F of this Report.

[33] Roskill Information Services Ltd., *The Economics of Asbestos*, pp. 8–10.

[34] Vagt, *Asbestos*, p. 7; and Roskill Information Services Ltd., *The Economics of Asbestos*.

[35] Hodgson, "Chemistry and Physics of Asbestos," pp. 68–73.

The commercial types of asbestos are not always mineralogically pure. One type of asbestos may be contaminated by another (for example, Quebec chrysotile is contaminated by deposits of tremolite). Alternatively, one type of asbestos may contaminate a non-asbestos mineral (for example, asbestos fibres have been found in a talc mine near Madoc, Ontario; tremolite and chrysotile are found in some talc deposits in the United States; and actinolite occurs in some deposits of vermiculite).[36]

B.5 Naturally Occurring Deposits

Apart from deposits that permit commercial exploitation, asbestos is ubiquitous in the crust of the earth and its surface waters. Serpentine minerals are found in the rocks of every major mountain chain in the world and in pre-Cambrian shield areas. Although concentrations large enough to mine are rare, these rocks will almost certainly contain some chrysotile asbestos. Thus, naturally occurring chrysotile will be found in soils, streams, lakes, and oceans in many localities.[37] The amphibole minerals also occur in a wide variety of rocks throughout the world, but most are in non-fibrous form.[38] The asbestiform amphiboles have a much more restricted occurrence.[39]

In summary, then, the deposits of asbestos can be divided into three categories: (i) deposits which are themselves commercially exploitable; (ii) deposits occurring in the rocks of mines developed primarily for the exploitation of other minerals; and (iii) natural occurrences found in many geological formations throughout the world.

C. The Commercial Production, Processing, and Uses of Asbestos

Reports of man's earliest use of asbestos date to the Stone Age, when it was utilized to strengthen clay pottery.[40] From that time forward, asbestos was used intermittently and in small amounts until the latter part of the nineteenth century. Canada's first asbestos mine opened in 1877 in Quebec, and the subsequent exploitation of these chrysotile deposits marked the commencement of large-scale asbestos mining and commercial production in that province.[41]

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[36] Chatfield, "The Problems of Measurement of Asbestos," Appendix A, p. 3; RCA Transcript, Evidence of Mr. Geoffrey Berry, 19 June 1981, Volume no. 11, p. 6; and Richard A. Lemen, moderator, "Discussion of Session V: Talcs Contaminated with Other Minerals," in *Dusts and Disease*, eds. Richard A. Lemen and John M. Dement (Park Forest South, Illinois: Pathotox Publishers, Inc., 1979), pp. 341–355.

[37] RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 114–115.

[38] For example, there is a deposit of non-asbestiform tremolite near Sharbot Lake, Ontario. See Chapter 6, Section F of this Report.

[39] Fred J. Wicks, "Mineralogy, Chemistry and Crystallography of Chrysotile Asbestos," in Mineralogical Association of Canada, *Short Course in Mineralogical Techniques of Asbestos Determination*, p. 36; and Zussman, "The Mineralogy of Asbestos," p. 56.

[40] U.K., Advisory Committee on Asbestos, *Asbestos — Volume I: Final Report of the Advisory Committee*, paragraph 3, p. 11.

During the next fifty years, there was a gradual increase in world production and use, with a cumulative total of just under 5 million tons having been mined by 1930. In the years following 1930, and particularly during and after World War II, world production accelerated dramatically, nearly doubling between 1960 and 1973 alone.[42] This huge increase was largely attributable to the extensive use of asbestos as a fireproofing and insulation material in ship construction during the war and in building construction from the 1950s until the early 1970s. Although in recent years production has levelled off as a result of increasing concern over health effects, discontinuation of certain uses of asbestos, and the development of effective substitutes, annual world asbestos production reached over 5 million metric tons by 1979.[43]

Canada was once the world's leading producer of asbestos, reaching its zenith in 1947 when it mined and milled two-thirds of the world's supply. Since that time, Canada's share of world production has decreased. In general, Canadian production levels have not changed significantly since 1965, and by 1975, Canada had been overtaken by the Soviet Union. Today, Canada remains the world's second largest producer of asbestos, supplying between 25 and 30% of world production. Figure 2.3 provides a breakdown of world asbestos production by country in 1979. Table 2.2 lists asbestos production for the three principal producing countries — Canada, South Africa, and the U.S.S.R. — for the period from 1948 to 1981.

Nearly 90% of Canadian production comes from Quebec and asbestos accounts for about one-third of the value of all mineral production in the province.[44] Ninety-five percent of total Canadian production is exported, making it the world's leading exporter. In 1978, Canadian exports amounted to over 1.4 million metric tons, having a value in excess of $600 million. The United States accounts for nearly 40% of total Canadian exports, and in turn, Canada supplies 99% of total United States imports of asbestos.[45]

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[41] Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 187.

[42] World Health Organization, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, vol. 14: *Asbestos*, pp. 29ff.

[43] Vagt, *Asbestos*, p. 7. According to *The Economics of Asbestos*, world consumption of asbestos increased from 4,231,000 metric tons in 1974 to 4,803,000 metric tons in 1980. However, in that period consumption fell 24% in North and Central America and 27% in Western Europe, while it rose 50% in South America, 86% in Eastern Europe, and 150% in Africa. Roskill Information Services Ltd., *The Economics of Asbestos*, p. 80.

[44] Stephen Shugar, *Effects of Asbestos in the Canadian Environment*, prepared for the NRC Associate Committee on Scientific Criteria for Environmental Quality, NRCC, no. 16452 (Ottawa: National Research Council Canada, 1979), p. 25.

Over 3,000 separate uses of asbestos have been identified in the literature. Of course, these uses depend principally upon the physical and chemical properties of asbestos fibres and the particular characteristics of individual fibre types. The following catalogue is illustrative only of the broad range of uses to which the properties of asbestos have enabled it to be put.[46]

Fire retardant — Asbestos is perhaps best known for its fire-resistant properties, and it has, for example, been widely utilized in the textile industry to increase resistance to heat and fire. Chrysotile in particular has been extensively used in textiles because its flexible and longer fibres can easily be carded, spun, and woven into yarn. Chrysotile asbestos also has been used to provide fire resistance in a wide variety of paper products.

Insulation — Asbestos is an effective insulator against heat, cold, electricity, and noise. Amosite and chrysotile in combination and, to a lesser extent, crocidolite were widely used in North America and elsewhere in sprayed insulation and fireproofing materials, especially in the shipbuilding industry during World War II and in the construction industry in the period immediately following the war until the early 1970s.

The heat-resistant qualities of amosite especially qualified it for these uses. The utilization of asbestos in ship and building insulation largely accounted for the huge increase in the demand for asbestos that began during the war. Thus, for example, asbestos was used as a thermal insulator in pipes and boilers, in the structural steelwork of highrise buildings, and also for acoustical and decorative purposes in ceiling tiles and building walls.[47] But these uses carried with them a serious health risk. The North American insulators, working with amosite and chrysotile asbestos, have one of the worst disease records of any group of employees that has been exposed to asbestos, a record first reported on in 1964 by Dr. Irving J. Selikoff and his

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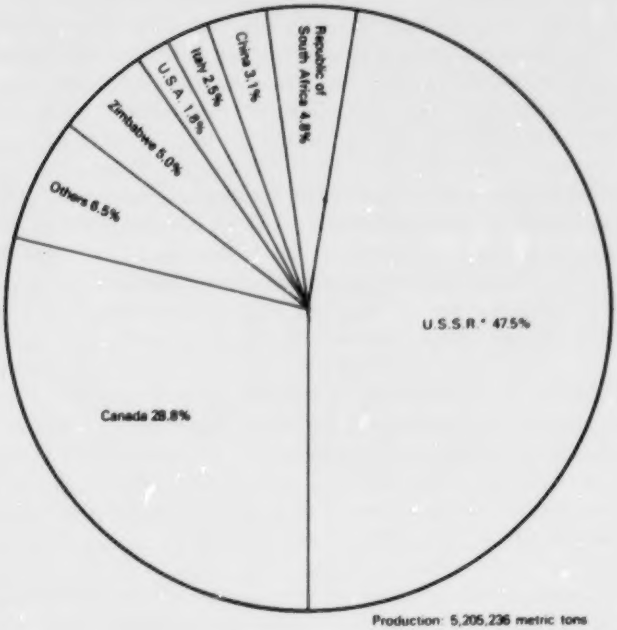
[45] Gouvernement du Québec, Ministère de l'Energie et des Ressources, Written submission to the Royal Commission on Asbestos, #19, January 1981, p. 4.

[46] See generally, Rajhans and Sullivan, *Asbestos Sampling and Analysis*, pp. 24ff.; Peters and Peters, *Sourcebook on Asbestos Diseases: Medical, Legal, and Engineering Aspects*, pp. A4–A7; and Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, *passim*.

[47] Irving J. Selikoff, "Asbestos-Associated Disease," in *Public Health and Preventive Medicine*, 11th ed., ed. John M. Last (New York: Appleton-Century-Crofts, 1980), chap. 13, p. 585.

Figure 2.3

World Asbestos Production by Country, 1979



Note: *Approximately equivalent to Canadian grades.

SOURCE: G. Oliver Vagt, *Asbestos* (Hull, Quebec: Supply and Services Canada, 1980), Figure 2, p. 7 (based on numerous sources).

Table 2.2

World Production of Asbestos, by Principal Producing Countries: 1948 to 1981 (000 metric tons)

	Canada		South Africa		U.S.S.R.		World Total
Year	000t	%	000t	%	000t	%	000t
1948	590	63	38	4	-	-	-
1949	473	53	58	7	-	-	-
1950	720	61	72	6	-	-	-
1951	801	62	88	7	-	-	-
1952	765	59	110	9	-	-	-
1953	827	58	86	6	272	19	1,420
1954	838	55	99	7	304	23	1,515
1955	965	55	109	6	408	23	1,755
1956	920	52	124	7	434	25	1,970
1957	949	50	143	8	434	24	1,887
1958	839	45	159	9	499	27	2,055
1959	953	46	165	8	544	26	2,059
1960	1,015	46	160	7	599	27	2,214
1961	1,065	42	177	7	798	32	2,513
1962	1,103	40	201	7	998	36	2,742
1963	1,157	46	187	8	685	27	2,505
1964	1,288	47	196	7	735	27	2,768
1965	1,259	44	218	8	785	28	3,146
1966	1,342	44	251	8	839	28	3,047
1967	1,317	45	244	8	769	26	2,910
1968	1,370	46	236	8	800	27	2,965
1969	1,431	44	258	8	962	30	3,265
1970	1,507	43	287	8	1,096	31	2,490
1971	1,483	41	322	9	1,152	32	3,585
1972	1,530	41	323	9	1,220	32	3,774
1973	1,690	43	334	8	1,280	31	4,179
1974	1,644	38	333	8	1,360	32	4,274
1975	1,056	26	355	9	1,650	41	3,992
1976	1,536	32	367	8	1,850	39	4,787
1977	1,517	32	380	8	1,900	40	4,793
1978	1,422	30	257	5	1,945	42	4,673
1979	1,493	30	249	5	2,100	42	4,992
1980	1,323	27	277	6	2,250	45	4,988
1981	1,133	24	273	6	2,340	49	4,817

SOURCE: Roskill Information Services Ltd., *The Economics of Asbestos*, 4th ed. (London, England: Roskill Information Services Ltd., 1983), Table 6, p. 9.

colleagues at Mount Sinai Hospital in New York.[48] Moreover, the asbestos-sprayed materials themselves flaked from building pipes and tiles with constant wear. Consequently, building occupant exposure to asbestos, however minimal, became a concern. Sprayed insulation materials were effectively banned in the United States in 1973 and their use was discontinued in Ontario in the same year.[49]

Friction and Wear — Because of its heat resistance and other qualities, chrysotile is extensively used in friction products, including linings for disc and drum brakes and clutch facings.

Reinforcement — Chrysotile, because of its very high tensile strength, resistance to alkalies, and flexibility, is used as a reinforcing agent in the asbestos-cement industry, whose products include asbestos-cement pipe, shingles, clapboards, and flat and corrugated sheets.[50] Because it enhances the reinforcement, the dispersion of the fibre, and the drainage properties of the asbestos-cement mix, limited amounts of crocidolite are used in this industry as well, particularly for the manufacture of asbestos-cement pressure pipe.[51]

Wear Resistance — Asbestos is employed in vinyl floor tiles providing excellent resistance to wear and tear, impermeability to water, strength, and fire resistance.

Resistance to Acids and Alkalies — Due to its excellent resistance to the corrosive action of acids and alkalies, crocidolite has been used mainly in the manufacture of battery boxes, packings, acid pumps, valves, and gaskets.[52]

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[48] Irving J. Selikoff, Jacob Churg, and E. Cuyler Hammond, "Asbestos Exposure and Neoplasia," *Journal of the American Medical Association* 188 (1964): 22–26. Idem, "The Occurrence of Asbestosis Among Insulation Workers in the United States," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 139–155; and Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943–1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 91–116.

[49] The stringent control requirements prescribed by Regulation 419/73 made under *The Construction Safety Act* forced their discontinuance in Ontario. See Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, p. 16. In the United States, the Environmental Protection Agency in 1973 prohibited "visible emissions" of asbestos from any work site. This effectively abolished the spraying of asbestos materials for fireproofing or decorative purposes. See William J. Nicholson, "Regulatory Actions and Experiences in Controlling Exposure to Asbestos in the United States," *Annals of the New York Academy of Sciences* 329 (26 October 1979): 293–303.

[50] Zielhuis, *Public Health Risks of Exposure to Asbestos*, p. 20.

[51] Ibid., p. 25.

[52] Ibid.

Filtering — During World War II, both crocidolite and chrysotile were used as the filter material in gas mask cannisters.[53] Asbestos fibres are also used today as a filter medium in the chemical, drug, and food industries.

Cohesion — The addition of small amounts of asbestos to asphalt-surfaced roadways has served to increase the cohesion and wear resistance of the pavement.

Filler — Asbestos is used as a filler in resins, plastics, caulking, and sealants.

Some of the individual processes that utilize asbestos are quite different and these differences may be significant in terms of health effects. All forms of manufacturing using asbestos are likely to involve opening bags and then emptying them into a hopper where the asbestos is mixed with other material. A good deal of manufacturing, including, for example, the manufacture of friction products, encompasses a step where the asbestos, mixed with binders and other materials, is ground up. By contrast, in textile spinning and weaving, the asbestos is formed into yarn, and the yarn is then moved at high speed over pulleys. These different processes may cause the release of fibres of different dimensions and hence differences in health effects, a subject we consider in detail in Chapter 5 of this Report.

We describe Canadian production and consumption of asbestos in Chapter 6 of this Report.

D. Asbestos-Related Diseases

Since at least the beginning of this century, medical and scientific literature has documented the health hazards resulting from the inhalation of asbestos dust. As we began our inquiry, it was a well-accepted fact that asbestos exposure can cause and has indeed caused serious and debilitating disease and often death. In this section we consider the diseases associated with asbestos exposure, namely, asbestosis, mesothelioma, carcinoma of the lung (commonly referred to as lung cancer), and other asbestos-related cancers. Each of these diseases is characterized by a period of latency: that is, the existence of a time interval, usually of many years, between first exposure to asbestos and the clinical manifestation of the disease.[54] Figure 2.4 depicts the principal asbestos-related diseases and conditions and their sites in the human body.

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[53] Alison D. McDonald and J. Corbett McDonald, "Mesothelioma After Crocidolite Exposure During Gas Mask Manufacture," *Environmental Research* 17 (December 1978): 340–346; and J.S.P. Jones et al., "The Consequences of Exposure to Asbestos Dust in a Wartime Gas-Mask Factory," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 637–653.

D.1 Asbestosis

Asbestosis is a chronic, restrictive lung disease due to the inhalation of asbestos fibres. It is characterized by diffuse interstitial fibrosis or scarring which produces a "small, tight lung." The most prominent symptom is breathlessness and a characteristic physical sign is the presence of crepitations (rales) or crackling noises in the lung. Clubbing or thickening of the fingers and toes may be present in cases of advanced disease. Typical pulmonary function changes include restriction of lung volume, diminished forced vital capacity, and a decrease in diffusion capacity. Characteristic x-ray changes of asbestosis are linear and irregular small opacities in the lower and middle lung zones.[55]

There is an incomplete correlation between the extent of x-ray abnormality (and in some cases measured lung function defects) and clinical well-being. Some patients whose x-rays show extensive scarring are able to work without difficulty and live without restraint, and the opposite is also true. There is some evidence to indicate that lung function changes are often a better early detector of asbestosis than x-ray changes, but this is not invariably the case.[56]

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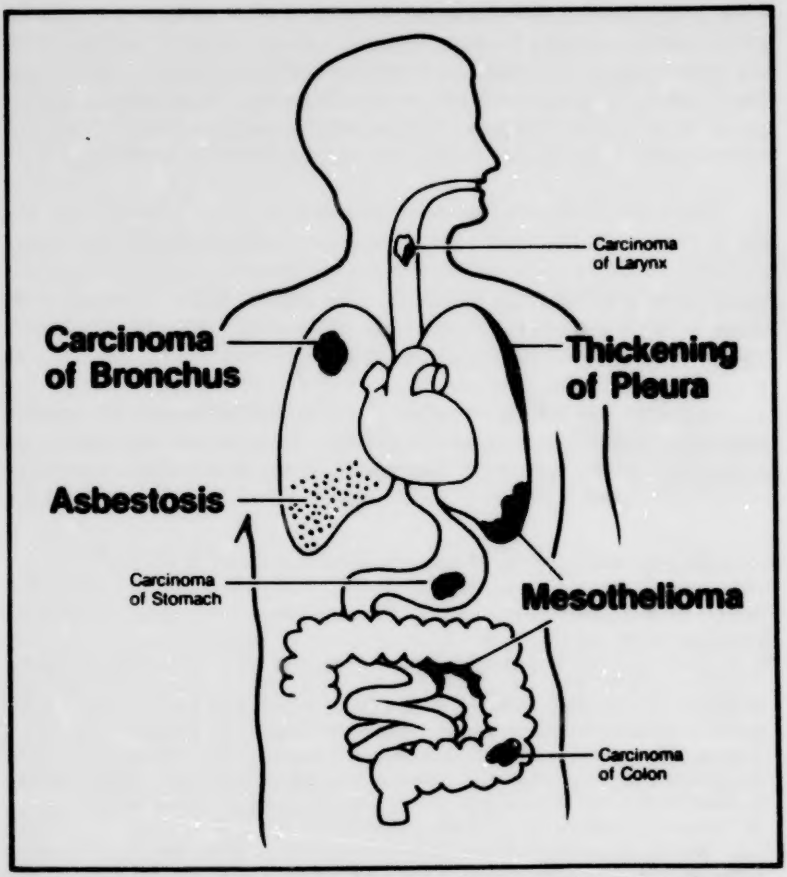
[54] See generally, Selikoff and Lee, *Asbestos and Disease*; and Selikoff, "Asbestos-Associated Disease," pp. 568–598. In the brief discussion that follows, no attempt is made to distinguish among different fibre types. The question of the relative pathogenicity of the various fibre types will be left to Chapter 5 of this Report.

[55] Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," pp. 205–206; RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, pp. 43–44; U.S., National Institute for Occupational Safety and Health, *Revised Recommended Asbestos Standard*, prepared by Richard A. Lemen and John M. Dement (Washington, D.C.: U.S. Department of Health, Education and Welfare, December 1976), pp. 29–30; RCA Transcript, Evidence of Dr. Alexander C. Ritchie, 14 July 1982, Volume no. 49, p. 63; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 55–56. With silicosis, and other fibrotic lung disease, the opacities are small and rounded. Often there will be exposure to both silica and asbestos.

[56] Selikoff, "Asbestos-Associated Disease," pp. 571ff.; and Hans Weill et al., "Lung Function Consequences of Dust Exposure in Asbestos Cement Manufacturing Plants," *Archives of Environmental Health* 30:2 (February 1975): 95. Dr. Hans Weill examined the lung function consequences of dust exposure in two asbestos-cement manufacturing plants in the New Orleans area. His findings indicated that individuals may have a reduction in pulmonary function prior to the time when x-ray abnormalities appear.

Figure 2.4

Principal Asbestos-Related Diseases and Conditions and Their Sites in the Human Body


Carcinoma of Larynx
Carcinoma of Bronchus
Thickening of Pleura
Asbestosis
Carcinoma of Stomach
Mesothelioma
Carcinoma of Colon

SOURCE: Illustration by Mr. Jerry Farrell, Audio-Visual Centre, McMaster University; consultative assistance by Dr. David C.F. Muir, Director, Occupational Health Program, Health Sciences Centre, McMaster University, Hamilton, Ontario.

Asbestosis has historically been associated with prolonged and heavy occupational exposure to asbestos.[57] The pulmonary fibrotic changes develop slowly over the years, are irreversible, and will normally progress even in the absence of further exposure to asbestos.[58] The disease can become seriously disabling. Asbestosis is, however, more often a cause of morbidity (illness) among asbestos workers than it is a cause of mortality. Still, those with asbestosis are susceptible to death from related causes — often infection or cardio-respiratory complications. For example, pulmonary hypertension is frequently associated with advanced asbestosis and the resultant right-sided heart failure (*cor pulmonale*) may be the stated cause of death.[59]

There is no effective treatment for asbestosis itself, which is irreversible. However, the physician's improved ability to treat some of the symptoms and secondary effects of the fibrosis has somewhat bettered the quality of life of patients with asbestosis. Present-day medical therapy now makes it possible to give the asbestotic some relief from the discomfort associated with complications arising from asbestosis.[60]

It is clear that individuals afflicted with asbestosis have an appreciable reduction in their life expectancy. Mr. Geoffrey Berry calculated that persons given a 10% disability rating for asbestosis by the British Pneumoconiosis Medical Panel had a reduction in life expectancy of three years, rising to twelve years for those 50% or more disabled.[61] In 1981, Dr. Murray M. Finkelstein, a medical consultant with the Ontario Ministry of Labour, reported on the mortality experience of 172 workers who had been awarded compensation for asbestosis by the Workers' Compensation Board. Not only did he find that these workers had increased rates of death compared to the general population for non-malignant respiratory disease, lung cancer, and mesothelioma, but he also found that in comparison with the general population, the proportion of workers who survived was only 69% five years after being awarded compensation, and only 53% after ten years.[62]

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[57] RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 52; RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, p. 96; Selikoff and Lee, *Asbestos and Disease*, p. 266; and Zielhuis, *Public Health Risks of Exposure to Asbestos*, p. 78.

[58] RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 12–13; RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 123; and Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 209. In her evidence, Dr. Margaret R. Becklake suggested that with certain fibrotic diseases there is a stage of activity at which intervention may prevent progression to fibrosis. But she thought it unlikely that it would ever be possible to identify such a stage in the asbestos-exposed individual. See RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, pp. 43–44.

[59] U.S., NIOSH, *Revised Recommended Asbestos Standard*, pp. 29–30; Selikoff, "Asbestos-Associated Disease," pp. 571–575; RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, pp. 69–70; RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 27; RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 136; RCA Transcript, Evidence of Dr. Alexander C. Ritchie, 14 July 1981, Volume no. 49, pp. 46–47; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 42, 126–127. As Dr. Weill testified, a person with asbestosis will have less lung reserve to cope with a serious infection such as pneumonia. In addition, fairly advanced asbestosis produces changes in pulmonary circulation that in turn put a strain on the heart. Moreover, it is possible that advanced asbestosis could preclude operations in respect of other diseases.

[60] Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 209; RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, p. 44; RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), pp. 79–80; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, p. 46.

D.2 Mesothelioma

Mesothelioma is a rather rare cancer arising from the surface-lining cells of the pleura and the peritoneum. These surface-lining cells are called the mesothelium and hence the name of the disease, mesothelioma. Malignant mesothelioma is of two types, pleural and peritoneal, depending upon whether the site of the tumour is the lung or the abdomen. The tumours that appear are unusual and striking. They are generally diffuse, spreading rapidly and widely over the large surfaces of the thoracic or abdominal cavities.[63]

There is no effective treatment for mesothelioma. Chemotherapy, immunology, radiation, and various forms of surgery have all produced disappointing results. At best, treatment programmes provide some brief measure of palliative relief or some short-term prolongation of what is certainly a very miserable life.[64] As Dr. Paul Kotin bluntly testified, for mesothelioma, morbidity and mortality are in practical terms identical. A large proportion of mesothelioma patients die within a year of diagnosis, and few survive longer than five years.[65] This bleak picture serves to emphasize that mesothelioma, like the other asbestos-related diseases, is a disease for which the prevention is far more effective than the cure.

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[61] Geoffrey Berry, "Mortality of Workers Certified by Pneumoconiosis Medical Panels as Having Asbestosis," *British Journal of Industrial Medicine* 38 (1981): 135; and RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, pp. 37–38.

[62] Murray M. Finkelstein, Robert A. Kusiak, and George Suranyi, "Mortality Among Workers Receiving Compensation for Asbestosis in Ontario," *Canadian Medical Association Journal* 125 (1 August 1981): 259–262.

[63] Selikoff, "Asbestos-Associated Disease," p. 580.

[64] Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 213; RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 135–137; and P. Ruffié and A. Hirsch, "A Review of the Treatment of Diffuse Malignant Pleural Mesothelioma," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 553–557.

[65] RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), p. 81; Selikoff, "Asbestos-Associated Disease," p. 583; and Selikoff and Lee, *Asbestos and Disease*, pp. 302–306.

Historically, the incidence of mesothelioma has been extremely high among three occupational groups exposed to asbestos: insulators, those who work in asbestos plants, and those who work in shipyards. But cases of mesothelioma have been known to occur not only among those occupationally exposed to asbestos but also among persons living in the same household as an asbestos worker and among persons living in the neighbourhood of asbestos mining or manufacturing activities.[66] Since 1966, the incidence of mesothelioma in Canada has been on average 31 cases per year.[67]

Mesothelioma was for a time thought to be uniquely associated with asbestos exposure. On the basis of the available data, there is still little doubt that the great majority of reported cases of mesothelioma are attributable to asbestos exposure.[68] While it is true there is now a growing body of evidence which strongly suggests that mesothelioma can occur and indeed has occurred in the absence of any known or likely exposure to asbestos, these cases are, comparatively, exceedingly rare.

The evidence that mesothelioma can be caused without asbestos exposure comes principally from three sources. First, there have been a number of case reports of mesothelioma from various locales where there was simply no possibility of asbestos exposure. Chief among them is the recently reported outbreak of mesothelioma in rural Turkey where there is no industry and no asbestos. These cases have been specifically attributed to exposure to fibrous zeolites found in the region.[69] Second, animal experimental work has demonstrated that glass and other man-made mineral fibres are capable of inducing mesothelioma in rats at rates which closely approximate those for asbestos.[70] Third, in a number of epidemiological surveys of mesothelioma, there are some cases for which there is no known history of asbestos exposure, be it occupational, domestic, or neighbourhood, and for which there is no evidence of asbestos involvement in the lung. Dr. J. Corbett McDonald and Dr. Alison D. McDonald, in their survey paper, "Epidemiology of Mesothelioma from Estimated Incidence," examined over 4,500 fatal cases of mesothelioma reported from 22 countries in the years from 1959 to 1976. In 923 cases, accounting for 38% of the cases from non-occupational studies with a recorded history, the authors were unable to find any definite or even probable history of asbestos exposure.[71] Similarly, Mr. Julian Peto and his colleagues, in a study of mesothelioma deaths in the Los Angeles area of California in the years from 1972 to 1979, could not establish evidence of any asbestos exposure in nearly one-third of the 188 total cases that occurred and in well over half of the female victims.[72] These statistics must be put in context. The numerically few cases of mesothelioma among those with no likely asbestos exposure are drawn from the entire population[73] and are therefore an infinitesimally small fraction of the cases of death in this population. By contrast, the much larger number of cases of mesothelioma with known or probable asbestos exposure is a noticeable fraction of a much smaller asbestos-exposed population.

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[66] See J. Corbett McDonald and Alison D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," *Preventive Medicine* 6:3 (September 1977): 426–446; Selikoff and Lee, *Asbestos and Disease*, Table 10-5, pp. 267–270 and Table 11-6, p. 293; Henry A. Anderson and Irving J. Selikoff, "Asbestos-Associated Radiographic Changes Among Household Contacts of Amosite Asbestos Workers," in *Induced Disease: Drug, Irradiation, Occupation*, ed. Leslie Preger (New York: Grune and Stratton, 1979), pp. 253–273; and Muriel L. Newhouse and Hilda Thompson, "Epidemiology of Mesothelial Tumors in the London Area," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 579–588.

[67] J.C. McDonald and A.D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," p. 431. Between 1966 and 1975, the annual incidence of malignant mesothelioma per million males aged 15 and over was 4.9 in Quebec, 2.6 in Ontario, and 2.1 in the rest of Canada. However, misdiagnosis, or more accurately over-diagnosis, may have been a factor: the corresponding figures for accepted incidence after approval of diagnosis by a recognized panel of Canadian pathologists were 2.4, 2.0, and 1.5 respectively. See also, Alison D. McDonald, "Mesothelioma Registries in Identifying Asbestos Hazards," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 447; and RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, p. 15. For a fuller discussion of the difficulties of diagnosing mesothelioma, see Chapter 4, Section D.2 of this Report.

[68] See J.C. McDonald and A.D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," p. 439; and Alison D. McDonald and J. Corbett McDonald, "Malignant Mesothelioma in North America," *Cancer* 46:7 (1 October 1980): 1650–1656.

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[69] See Y. Izzettin Baris, Mustafa Artvinli, and A. Altay Sahin, "Environmental Mesothelioma in Turkey," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 423–432; and Fred D. Pooley, "Evaluation of Fibre Samples Taken from the Vicinity of Two Villages in Turkey," in *Dusts and Disease*, pp. 41–44. The data come from the villages of Karain and Tuzköy in Turkey. In the village of Karain in the years from 1970 to 1974, with a population between 600 and 800, there were a total of 24 pleural mesotheliomas. In 1974 alone, 11 of the 18 deaths in the village were from this tumour. The fibrous zeolite mineral erionite has been particularly implicated.

[70] And, as with asbestos, it is the longer and thinner man-made mineral fibres which appear to be more pathogenic in relation to tumour induction in animals. See Mearl F. Stanton and Constance Wrench, "Mechanisms of Mesothelioma Induction with Asbestos and Fibrous Glass," *Journal of the National Cancer Institute (JNCI)* 48:3 (March 1972): 797–821; and Mearl F. Stanton and Maxwell Layard, "The Carcinogenicity of Fibrous Minerals," in *Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods*, pp. 143–150. The issue of fibre dimension is discussed in Chapter 5, Section D of this Report.

[71] J.C. McDonald and A.D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," p. 429. The authors identified 268 cases from occupational studies, all with asbestos exposure; the authors also identified 4,271 cases from other studies, of which they had a recorded history for 2,453 cases. Of these 2,453 cases, 923 had no definite or probable history of asbestos exposure.

[72] Julian Peto, Brian E. Henderson, and Malcolm C. Pike, "Trends in Mesothelioma Incidence in the United States and the Forecast Epidemic Due to Asbestos Exposure During World War II," in *Banbury Report 9: Quantification of Occupational Cancer*, eds. Richard Peto and Marvin Schneiderman ([Cold Spring Harbor, New York]: Cold Spring Harbor Laboratory, 1981), pp. 51–69; and RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 133.

Accordingly, in our view, mesothelioma remains a disease that is quite specifically linked to asbestos exposure. While we cannot conclude that asbestos exposure is the sole cause of mesothelioma and cannot discount the possibility that present and future industrial processes using non-asbestos fibres will produce mesotheliomas, we can state that the possibility that an individual without asbestos exposure will contract the disease is remote.[74]

D.3 Lung Cancer

Lung cancer, unlike asbestosis or mesothelioma, is not specifically associated with asbestos exposure. Moreover, it has had a particular history of association with cigarette smoking.[75] Pathologically, there does not appear to be any basic difference between pulmonary carcinoma in general and that associated with persons exposed to asbestos, save that whereas the majority of tumours appear in the upper portion of the lungs, those in asbestos-exposed individuals appear more commonly (but not always) in the lower lobes.[76] However, serious issues of causation have been raised when lung cancer develops in asbestos workers, especially in the absence of co-existing asbestosis. While most persons exposed to asbestos who develop lung cancer are smokers, it is now generally recognized that asbestos on its own in the absence of smoking is capable of inducing lung cancer and that the tumour may develop even where there is no co-existing asbestosis.[77] The relationship between asbestos, cigarette smoking, and human diseases will be explored further in Chapter 5 of this Report.

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[73] More accurately, this means the entire non-asbestos population. The McDonald and McDonald survey included 22 countries. Before these statistics can be accepted at face value, two observations need be made: first, it is possible that the histories of those patients for whom there is said to be no asbestos exposure is inaccurate because such exposure has occurred without their knowledge or the knowledge of those interviewed; second, mesothelioma was and continues to be a difficult diagnosis and the possibility for misdiagnosis is considerable. These observations do perhaps cast some doubt on the accuracy of the mesothelioma surveys, but it is not warranted in our view to discount in their entirety the numerous reported cases with no demonstrable asbestos exposure in light of the consistency of such a finding in the various groups of cases that have been studied.

[74] It is in fact so rare that there is no calculable background incidence rate for mesothelioma in the general population.

[75] E. Cuyler Hammond, Irving J. Selikoff, and Herbert Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 473–490.

[76] RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, p. 78.

The prognosis and treatment of lung cancer in those exposed to asbestos are little different from lung cancer in the general population. Only about one in twenty persons with lung cancer survives longer than five years from the diagnosis of the disease.[78] If asbestosis is present, the prospects for a person with an asbestos-related lung cancer could be worse because the asbestosis may limit the treatment options available to the physician and may in particular preclude surgical intervention.[79]

D.4 Other Asbestos-Related Cancers

Other malignant neoplasms have also come to be associated with the inhalation of asbestos. An excess risk of gastrointestinal cancer affecting all sites in the gastrointestinal tract (esophagus, stomach, colon, and rectum) has been demonstrated in a number of studies of asbestos workers.[80] More recently, certain studies have also implicated asbestos as a cause of cancer of the larynx.[81] However, the association between asbestos exposure and both gastrointestinal cancer and cancer of the larynx is neither as strongly nor as consistently established in the medical literature as the association between asbestos exposure and the diseases previously discussed.[82]

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[77] Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 214; Selikoff and Lee, *Asbestos and Disease*, p. 309; RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), p. 76 and 23 July 1981, Volume no. 21(B), pp. 19-20; Hammond, Selikoff, and Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," pp. 485-488; and J. Corbett McDonald, "Asbestos and Lung Cancer: Has the Case Been Proven?" *Chest* 78:2 (August 1980, Supplement): 374-376.

[78] RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), p. 81; and Selikoff, "Asbestos-Associated Disease," p. 575.

[79] Asbestosis may limit the treatment options for other diseases as well; see, for example, the case of Mr. John Dodds, referred to in Chapter 13, Section C.1 of this Report. See Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 214.

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[80] Selikoff, "Asbestos-Associated Disease," pp. 583–584; and Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 214. Both Dr. Alexander C. Ritchie and Dr. Anthony B. Miller reviewed the evidence pertaining to asbestos exposure and gastrointestinal cancer for the Ontario Workers' Compensation Board. Dr. Ritchie in 1976 found there was little non-epidemiological evidence to support the suggestion that exposure to asbestos increases the risk of developing carcinoma of the gastrointestinal tract. His conclusion that asbestos exposure increases the risk of developing carcinoma of the colon and rectum and probably of the esophagus and stomach rests almost entirely on the epidemiological evidence and principally on the work of Dr. Selikoff and his colleagues. Dr. Miller concluded that there is no doubt that an association has been demonstrated for all sites in the gastrointestinal tract, but that the demonstrated association is weak. See The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, p. 27. Dr. E. Donald Acheson and Dr. Martin J. Gardner, in their report to the U.K. Advisory Committee on Asbestos, expressed reservations as to the association. In his testimony before us, Dr. Acheson was of the view that the evidence since the 1979 publication of his co-authored report of an association between asbestos exposure and gastrointestinal cancer was if anything a little weaker, and he suggested the possibility of another factor at work. See E. Donald Acheson and Martin J. Gardner, "The Ill Effects of Asbestos on Health," in U.K., Advisory Committee on Asbestos, *Asbestos — Volume 2: Final Report of the Advisory Committee*, paragraphs 211-213, p. 41; and RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, pp. 6, 72.

[81] P.M. Stell and T. McGill, "Asbestos and Laryngeal Cancer," *The Lancet* 2:826 (25 August 1973): 416–417; R.W. Morgan and P.T. Shettigara, "Occupational Asbestos Exposure, Smoking, and Laryngeal Carcinoma," *Annals of the New York Academy of Sciences* 271 (28 May 1976): 308–310; and M.L. Newhouse, M.M. Gregory, and H. Shannon, "Etiology of Carcinoma of the Larynx," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 687–695. Both Dr. Alexander C. Ritchie and Dr. Anthony B. Miller reviewed the evidence on asbestos exposure and cancer of the larynx for the Ontario Workers' Compensation Board. See The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, pp. 31–32, 40, 42.

[82] An association between asbestos exposure and other cancers, for example, cancer of the kidney, has also been suggested but not satisfactorily demonstrated. See Selikoff, "Asbestos-Associated Disease," p. 584. In their update of "The Ill Effects of Asbestos on Health," Dr. E. Donald Acheson and Dr. Martin J. Gardner suggested a possible association between asbestos exposure (likely amphiboles) and ovarian cancer in women. See E. Donald Acheson and Martin J. Gardner, *Asbestos: The Control Limit for Asbestos*, prepared for the U.K. Health and Safety Commission (London: Her Majesty's Stationery Office, 1983), paragraph 17, p. 3.

D.5 Other Asbestos-Related Conditions

(a) Pleural Changes (pleural thickening, pleural effusions, and pleural plaques)

Asbestos exposure can also produce scarring, both localized and diffuse, of the lining of the pleural surfaces, giving rise to pleural thickening, benign pleural effusions, and pleural plaques.[83] Plaques are white, glistening, raised areas of fibrous tissues which may calcify or harden. In general, these limited pleural changes and plaques are not associated with clinical and functional abnormalities. They are simply markers of asbestos exposure. However, once in a while this pleural process can become rather diffuse and can severely restrict lung function, thereby incapacitating the individual.[84] Pleural changes may or may not be accompanied by asbestosis. Some schools of medicine, notably the Mount Sinai School of Medicine in New York, consider these pleural changes as "pleural asbestosis," but the term is not widely used, undoubtedly because these changes generally do not produce clinically disabling effects similar to asbestosis.[85]

(b) Asbestos Bodies and Warts

Certain particles of asbestos fibres, once inhaled in the lungs, may become coated with a substance containing protein and iron to form asbestos bodies. Fibres other than asbestos can provide a core on which similar coatings can develop and therefore the generic term "ferruginous bodies" is often used. Asbestos bodies are not themselves harmful but represent a benign tissue reaction to asbestos fibres. Principally they serve as evidence of asbestos exposure,[86] as do asbestos warts which are harmless skin growths that occur when asbestos fibres penetrate the skin.[87]

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[83] The plaques may be hyaline or calcified. See Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," pp. 200–202.

[84] RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 13–14; and RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, p. 66.

[85] RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, p. 14; and Selikoff and Lee, *Asbestos and Disease*, pp. 143ff. and especially pp. 189ff. The Mount Sinai School of Medicine in New York distinguishes between "pleural" and "parenchymal" asbestosis.

E. The Recognition of Asbestos as a Health Hazard and the Historical Incidence of Disease

It was the fibrogenic effects of asbestos inhalation that triggered the recognition of asbestos as a health hazard. The earliest known cases of asbestosis were among asbestos textile workers. For example, in 1906, M. Auribault, an inspector in the Department of Labour in France, reported some 50 deaths in the period 1890–1895 among workers at an asbestos weaving mill in Calvados.[88] In 1907, Dr. H. Montague Murray related the death of a patient from fibrosis of the lungs due to the inhalation of asbestos dust to a parliamentary committee on industrial disease compensation in Great Britain, the victim being a 33 year old man who had worked for 10 years in the carding room of an asbestos textile mill.[89] In 1912, a Canadian health inspector who had examined conditions in an asbestos factory and in asbestos mines and mills noted that large amounts of asbestos dust had a weakening effect on the lungs.[90] The insurance industry too recognized the health hazards of asbestos at an early stage. Dr. Frederick L. Hoffman, statistician and Vice-President of the Prudential Insurance Company, wrote in 1918 ". . . that in the practice of American and Canadian life insurance companies asbestos workers are generally declined on account of the assumed health-injurious conditions of the industry."[91] We note parenthetically that the practice referred to by Dr. Hoffman has not become a policy of insurance companies.[92]

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[86] Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," pp. 194ff.; and Canadian Centre for Occupational Health and Safety, *A Review of Four Major Reports on the Health Hazards of Asbestos*, Royal Commission on Asbestos Background Paper Series, no. 2 (Toronto: Royal Commission on Asbestos, 1981), p. 4. Asbestos bodies are found in the lungs of many urban residents. They are not harmful but are simply indicative of an environmental exposure to asbestos. It has been estimated that no more than 50% of all asbestos fibres in the lung form themselves into asbestos bodies. There is some evidence to suggest that the coating of asbestos fibres renders them at least non-fibrogenic. See, for example, RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 15.

[87] Gordon Atherley, "A Survey of Four Studies on the Health Effects of Asbestos," in Ontario, Royal Commission on Asbestos, *Proceedings of The Royal Commission on Asbestos, First Public Meeting, Friday, October 31, 1980*, reported by Elizabeth J. Hiscott (Toronto: Royal Commission on Asbestos, 1980), p. 25; and Zielhuis, *Public Health Risks of Exposure to Asbestos*, p. 81. Asbestos warts are generally found only in asbestos workers.

[88] M. Auribault, "Note sur l'hygiène et la sécurité des ouvriers dans les filatures et tissages d'amiante," *Bulletin de l'Inspection du Travail* 14 (1906): 126.

[89] H. Montague Murray, "Statement Before the Committee in the Minutes of Evidence," in *Report of the Departmental Committee on Compensation for Industrial Disease* (London: His Majesty's Stationery Office, 1907), pp. 127–128.

[90] "Effect of Asbestos Dust on Workers' Health in Asbestos Mines and Factories," *Labour Gazette*, Canada (February 1912): 761–762.

Dr. W.E. Cooke, in a note in the *British Medical Journal* in 1924, described the death from pulmonary fibrosis of a 33 year old woman who had worked in an asbestos textile factory for 20 years. In a further report on the same case in 1927, Dr. Cooke gave the disease its current name, asbestosis.[93] An investigation of the condition of textile factory workers was undertaken in Great Britain in 1928 and 1929, and the resulting report to Parliament in 1930 recognized "... the fact that the inhalation of asbestos dust over a period of years results in the development of a serious type of fibrosis of the lungs." The report recommended the remedy of dust suppression which led to the promulgation of the Asbestos Industry Regulations in 1931.[94] This was the first regulation of which we are aware that was specifically directed to the control of asbestos in the workplace.

In the United States, a series of clinical reports in the first part of the 1930s confirmed the frequent occurrence of asbestosis among asbestos workers.[95] These reports were followed by two industry-wide studies. In the first, undertaken by the Metropolitan Life Insurance Company at the request of industry, Lanza, McConnell, and Fehnel reported that two-thirds of the x-ray films of 126 persons selected more or less at random from workers with more than three years' employment in asbestos mines and mills in Quebec and in fabricating plants along the Atlantic coast were not normal.[96] In the second, Dreessen et al. reported in 1938 on findings among 541 workers in asbestos textile plants in North Carolina. Although the study was seriously flawed because it included many newly hired employees and did not fully take account of the workers who had already left the workplace due to disability, there were still numerous demonstrated cases of asbestosis, particularly among those most heavily exposed.[97]

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[91] Frederick L. Hoffman, "Mortality from Respiratory Diseases in Dusty Trades (Inorganic Dusts)," *Bulletin of U.S. Bureau of Labor Statistics, no. 231*, Industrial Accidents and Hygiene Series, no. 17 (Washington, D.C.: U.S. Bureau of Labor, June 1918), p. 178.

[92] Dr. Hoffman's observation received a great deal of attention in submissions to this Commission. See, for example, Ontario Federation of Labour, Written submission to the Royal Commission on Asbestos, #35, January 1981, p. 7; United Electrical, Radio and Machine Workers of America, Written submission to the Royal Commission on Asbestos, #36, January 1981, pp. 2-3; and Canadian Centre for Occupational Health and Safety, Written submission to the Royal Commission on Asbestos, #44, January 1981, p. 5. It may be that this observation had some historical validity. However, we are satisfied that it has not represented the practice of insurance companies conducting business in Canada for a very considerable period of time. Our staff made enquiries of eighteen insurers and in no case did an insurer indicate that an individual was denied coverage because he was an asbestos worker. Generally, insurers indicated they do not impose extra mortality premiums for life insurance solely on the ground of employment in work involving exposure to asbestos. The health history of an individual asbestos worker may, however, result in higher premiums.

[93] W.E. Cooke, "Fibrosis of the Lungs Due to the Inhalation of Asbestos Dust," *British Medical Journal* 2 (1924): 147; and W.E. Cooke, "Pulmonary Asbestosis," *British Medical Journal* 2 (3 December 1927): 1024. See also, the discussion in Selikoff and Lee, *Asbestos and Disease*, pp. 22ff.

[94] E.R.A. Merewether and C.W. Price, *Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry* (London: His Majesty's Stationery Office, 1930); and U.K., Asbestos Industry Regulations (1931), Statutory Rules and Orders, 1931, no. 1140 (London: His Majesty's Stationery Office, 1931).

On the compensation side, in the United States, the first formal claim for compensation associated with asbestos exposure was made in 1927. In 1933, Johns-Manville Corporation, even then a leading asbestos producer, settled 11 asbestosis claims out of court for a total sum of $35,000.[98] Compensation for disease resulting from asbestos became possible in Ontario in 1926, when pneumoconiosis was recognized by *The Workmen's Compensation Act*;[99] and in 1942, the Board allowed its first claim for asbestosis.[100] Thus, it could be said that by the time of World War II, the hazard of asbestos as a pneumoconiotic dust was generally recognized.[101]

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[95] See, for example, K.M. Lynch and W.A. Smith, "Asbestos Bodies in Sputum and Lung," *Journal of the American Medical Association* 95 (30 August 1930): 659–661; R.G. Mills, "Pulmonary Asbestosis — Report of a Case," *Minnesota Medical Journal* 13 (1930): 495–499; J. Donnelley, "Pulmonary Asbestosis," *American Journal of Public Health* 23 (December 1933): 1275–1281; and P. Ellman, "Pulmonary Asbestosis: Its Clinical, Radiological, and Pathological Features, and Associated Risk of Tuberculosis Infection," *Journal of Industrial Hygiene* 15 (July 1933): 165–183.

[96] A.J. Lanza, W.J. McConnell, and J.W. Fehnel, "Effects of the Inhalation of Asbestos Dust on the Lungs of Asbestos Workers," *Public Health Reports* 50:1 (4 January 1935): 1–12.

[97] Waldemar C. Dreessen et al., *A Study of Asbestosis in the Asbestos Textile Industry*, U.S. Public Health Bulletin, no. 241 (Washington, D.C.: U.S. Government Printing Office, August 1938), pp. 68–69. Only 13% of the group surveyed had 10 or more years' employment and only 3 persons had worked more than 20 years. Moreover, it was later reported by Lynch and Ayer of the U.S. Public Health Service that ". . . the incidence of frank disease was so great that prior to the beginning of the study, out of a total of less than 600 employees, the plants discharged 150 workers suspected of having asbestosis." Only a portion of this group was examined. See Nicholson, "Regulatory Actions and Experiences in Controlling Exposure to Asbestos in the United States," p. 293.

[98] *Asbestos Litigation Reporter*, 7 February 1979.

[99] Pneumoconiosis was added to Schedule 3 by amendment to Regulation 94, 1 June 1926.

[100] The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, p. 14.

[101] *Borel v. Fibreboard Paper Products Corporation* at 1083, where the court stated in 1973 that "Asbestosis has been recognized as a disease for well over fifty years. . . . By the mid-1930s, the hazard of asbestosis [sic] as a pneumoconiotic dust was universally accepted."

There are two observations we might make concerning the recognition and incidence of asbestosis. First, there was a decades-long delay between the time asbestos was first identified as being able to cause fibrosis and the time fibrosis was generally accepted as a health hazard of asbestos exposure. This delay was no doubt due at least in part to the latency period between first exposure and the clinical manifestation of the disease. Second, the available evidence strongly suggests that the historical incidence of asbestosis was associated with heavy dust exposures. There are many reports of the dusty conditions in asbestos plants in the 1920s and 1930s; ventilation and other dust suppression equipment was minimal or nonexistent.[102] In many instances, these dusty conditions persisted right through to the 1960s, and where this occurred the toll of victims of asbestosis remained high.[103]

While the earlier recognition of the disease of asbestosis signalled the dangers of asbestos exposure, the later appraisal of factors causing cancer led to the identification of asbestos as a cause of this disease as well. Significantly, as historic exposure levels in workplaces have been reduced over time, cancer has overtaken asbestosis as the disease of concern resulting from asbestos exposure.

Attention was first called to the possible carcinogenic potential of asbestos in the mid-1930s with a series of medical reports both in the United States and in Great Britain associating asbestos exposure with the development of lung cancer.[104] These scattered case reports were augmented by the *Annual Report of the Chief Inspector of Factories for the Year 1947* in Great Britain. All of the cases known to the Inspectorate to have died from asbestosis since 1924 were reviewed, and more than 13% had lung cancer at a time when, in Great Britain, only 1% would have been expected to have the disease.[105] Then, in 1955, in a seminal epidemiological study, Sir Richard Doll documented that among a group of men employed 20 or more years at Rochdale, an asbestos textile plant in Northern England, the risk of lung cancer was increased tenfold.[106] Even so, Braun and Truan, in 1958, in a study for the Quebec Asbestos Mining Association, concluded there was no risk of cancer associated with asbestos exposure among the Quebec miners.[107] But, in the early 1960s, additional occupational studies provided extensive new evidence. For example, in 1963, Mancuso and Coulter confirmed Doll's findings of increased lung cancer mortality from asbestos exposure in a study of a United States asbestos manufacturing company.[108] In a study published in 1964, Selikoff, Churg, and Hammond found excess death rates from lung cancer among a group of New York - New Jersey insulators in the construction and shipbuilding trades to be 7 times expected rates.[109] By the time Dr. Selikoff and others had reported at the Conference on the Biological Effects of Asbestos organized by the New York Academy of Sciences at the end of 1964, few any longer disputed the association between asbestos exposure and carcinoma of the lung.[110] Subsequent studies, many of which will be considered in this Report, have amply confirmed this association.[111] But, as with asbestosis, so with lung cancer, the path from initial identification to full recognition of the association has taken decades.

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[102] For example, Dr. Irving J. Selikoff and Dr. Douglas H.K. Lee have stated that "Before 1930, conditions in the mines, mills, and factories were largely uncontrolled so that some heavy exposures occurred. Eyewitnesses speak of being unable to see more than a few feet, for instance, because of the amount of dust in the air." See Selikoff and Lee, *Asbestos and Disease*, p. 170. In his testimony before this Commission, Dr. J.C. McDonald described historical conditions in the Quebec mines in these terms: "[Y]ou couldn't see the workers . . . literally couldn't see them. They were in a cloud that you couldn't measure. . . . I'm not speaking of throughout, but I'm speaking particularly of the bagging department, where it was sometimes a habit of even getting into the bag and jumping on it, to pack the stuff in." RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 139.

[103] See Nicholson, "Regulatory Actions and Experiences in Controlling Exposure to Asbestos in the United States." Even with improvements in dust control, some textile operations remained very dusty. See Jeremiah R. Lynch and Howard E. Ayer, "Measurement of Dust Exposures in the Asbestos Textile Industry," *American Industrial Hygiene Association Journal* 27 (September/October 1966): 431–437; and Hilton C. Lewinsohn et al., "Dust Control in a Conventional Asbestos Textile Factory," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 225–241. Dust levels in Canadian mines and mills remained high into the 1950s. See G.W. Gibbs and R.S.J. du Toit, "Environmental Data in Mining," in *Biological Effects of Asbestos*, eds. P. Bogovski et al., IARC Scientific Publications, no. 8 (Lyon, France: International Agency for Research on Cancer, 1973), pp. 138–144.

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[104] K.M. Lynch and W.A. Smith, "Pulmonary Asbestosis III. Carcinoma of Lung in Asbesto-silicosis," *American Journal of Cancer* 14 (1935): 56–64; S.R. Gloyne, "Two Cases of Squamous Carcinoma of the Lung Occurring in Asbestosis," *Tubercle* 17 (October 1935): 5–10; S.R. Gloyne, "A Case of Oat Cell Carcinoma of the Lung Occurring in Asbestosis," *Tubercle* 18 (December 1936): 100–101; and D.S. Egbert and A.J. Geiger, "Pulmonary Asbestosis and Carcinoma, Report of a Case with Necropsy Findings," *American Review of Tuberculosis* 34 (July 1936): 143–146.

[105] See Selikoff and Lee, *Asbestos and Disease*, pp. 26ff.; Selikoff, "Asbestos-Associated Disease," pp. 569ff.; and E.R.A. Merewether, *Annual Report of the Chief Inspector of Factories for the Year 1947* (London: His Majesty's Stationery Office, 1947).

[106] Richard Doll, "Mortality from Lung Cancer in Asbestos Workers," *British Journal of Industrial Medicine* 12 (1955): 81–86.

[107] D.C. Braun and T.D. Truan, "An Epidemiological Study of Lung Cancer in Asbestos Miners," *Archives of Industrial Health* 17 (1958): 634–653.

[108] Thomas F. Mancuso and Elizabeth J. Coulter, "Methodology in Industrial Health Studies, The Cohort Approach, with Special Reference to an Asbestos Company," *Archives of Environmental Health* 6 (January–June 1963): 210–226.

[109] Selikoff, Churg, and Hammond, "Asbestos Exposure and Neoplasia."

[110] *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): *passim.*

[111] For a review of many of these studies, see Chapter 5, Section B of this Report.

In the 1940s and 1950s, there were scattered medical reports of the development of the third principal disease caused by asbestos exposure: mesothelioma. For example, 2 cases of mesothelioma were included in the 8 lung cancer cases found in the 4,000 asbestos miners in Quebec surveyed by Cartier in 1952.[112] Still, this tumour was thought to occur exceedingly rarely. Then, in 1960, Wagner, Sleggs, and Marchand reported 33 cases of pleural mesothelioma observed since 1956 in South Africa, of which 13 cases occurred among Cape crocidolite miners and 14 among persons living in the neighbourhood of these mines; and all but one had a proven association with asbestos exposure.[113] By the end of 1961, Dr. J. Christopher Wagner and his colleagues had diagnosed a total of 87 pleural and 2 peritoneal mesothelioma cases in South Africa, more than half of whom had never worked in the asbestos industry but had lived in the vicinity of the crocidolite mines and mills.[114] Since that time, the association between asbestos exposure and mesothelioma has been amply demonstrated in studies and case reports from many different countries.[115]

The studies by Dr. Selikoff and by Dr. Wagner were important not only in bringing to the attention of the public the association between asbestos exposure and cancer, but equally in pointing out that the risk of disease was not confined to asbestos workers in mining and manufacturing.

Dr. Selikoff's investigation of the mortality experience of insulators, which he extended to a North America-wide study, vividly showed for the first time the high incidence of asbestos-related disease outside fixed place industry.[116] It brought into view the fact that asbestos insulation workers and, by extension, demolition workers, building maintenance and repair personnel who might be exposed to asbestos during removal operations, and indeed the entire construction industry were at increased risk of asbestos-related disease. It has been estimated that two-thirds of all asbestos used in the United States between 1890 and 1970 was utilized in the construction industry.[117] Control procedures are inevitably more difficult outside fixed workplaces and have been slow in coming to the construction industry. Furthermore, exposures to asbestos, albeit often of short duration, were commonly intense when they occurred. In these circumstances, it is hardly surprising to find a high incidence of disease outside fixed place industry. Further concern about the incidence of disease outside asbestos mining and manufacturing was raised in 1968 when Harries reported 5 cases of pleural mesothelioma among civilian employees of the Royal Navy Devonport Dockyard in Plymouth, England.[118] By 1973, Harries had observed 55 cases of mesothelioma at Devonport, thus confirming shipyard exposures as an important source of asbestos-related disease.[119] Moreover, only 2 of the mesotheliomas found by Harries occurred in asbestos insulation workers; the remainder were among people in other trades — boilermakers, welders, etc. — who worked in shipyards together with insulation workers but did not themselves use asbestos often.[120] Their exposures to asbestos were at best intermittent or indirect. Following Harries' studies, similar findings were made of asbestos-related disease among shipyard workers employed during and after World War II in the United States.[121] Again, the time interval until clinical manifestation appears to have precluded an earlier discovery of this important source of disease.

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[112] P. Cartier, "Discussion," *Archives of Industrial Hygiene and Occupational Medicine* 5 (1952): 262.

[113] J.C. Wagner, C.A. Sleggs, and P. Marchand, "Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province," *British Journal of Industrial Medicine* 17 (1960): 260–271.

[114] J. Christopher Wagner, "Epidemiology of Diffuse Mesothelial Tumors: Evidence of an Association from Studies in South Africa and the United Kingdom," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 575–578.

[115] See Chapter 5 of this Report. See generally, J.C. McDonald and A.D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence"; and Selikoff and Lee, *Asbestos and Disease*, pp. 241–306.

[116] Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Cancer Risk of Insulation Workers in the United States," in *Biological Effects of Asbestos*, pp. 209–216; and Selikoff, Hammond, and Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943–1976."

[117] Selikoff, "Asbestos-Associated Disease," p. 585.

Dr. Wagner's observations of mesotheliomas occurring in the vicinity of the South African Cape crocidolite mines among persons who were not occupationally exposed suggested the potential importance of neighbourhood or environmental exposures in the development of this disease. In 1965, Newhouse and Thompson reported 11 cases of mesothelioma in England whose only known asbestos exposure arose from their living in the same household as asbestos workers, indicating yet another source of this disease.[122] Further cases of mesothelioma both from neighbourhood exposures and from family contacts have subsequently been reported.[123]

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[118] P.G. Harries, "Asbestos Hazards in Naval Dockyards," *Annals of Occupational Hygiene* 11:2 (1968): 138.

[119] P.G. Harries, "Experience with Asbestos Disease and Its Control in Great Britain's Naval Dockyards," *Environmental Research* 11:2 (April 1976): 261–267. These 55 cases occurred in the period 1964 to September 1973. There was one peritoneal mesothelioma. All the rest were pleural. Further, 156 men were awarded disability pensions for asbestosis in the period 1964–1973.

[120] Ibid., pp. 261–263. One was an asbestos sprayer and the other a lagger. Harries indicated that "Crocidolite asbestos was extensively applied by a spray process to deckheads and bulkheads for environmental insulation from 1940 to 1960. Records show that asbestos dust concentrations of 177–322 f/cc (fibers 2–10 m long) occurred during the spray process. . . ." (p. 261.) See also, Selikoff, "Asbestos-Associated Disease," p. 584.

[121] Irving J. Selikoff and E. Cuyler Hammond, "Asbestos-Associated Disease in United States Shipyards," *Ca — A Cancer Journal for Clinicians* 28:2 (March/April 1978): 87–89; Irving J. Selikoff, Ruth Lilis, and William J. Nicholson, "Asbestos Disease in United States Shipyards," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 295–311; Phillip L. Polakoff, Barry R. Horn, and Oscar R. Scherer, "Prevalence of Radiographic Abnormalities Among Northern California Shipyard Workers," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 333–339; and Jean Spencer Felton, "Radiographic Search for Asbestos-Related Disease in a Naval Shipyard," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 341–352.

F. The General Health Issues of Current Concern

The epidemiological findings in the 1960s evidencing the association between asbestos and cancer and suggesting that the risk of disease was not confined to asbestos workers served to heighten the public awareness of the health hazards of asbestos and intensify investigation of its ill effects. In the 1970s, there appeared a whole host of new epidemiological studies of defined populations exposed to asbestos in one way or another.[124] These studies certainly confirmed the findings of the 1960s, but they also raised new issues for consideration and discussion. There were, for example, certain studies which appeared to implicate the amphiboles as the more hazardous asbestos fibres, particularly in the production of mesothelioma.[125] Thus, fibre type became an issue, and the controversy was over whether chrysotile was less hazardous than amosite and crocidolite or equally hazardous, a controversy of considerable practical importance since approximately 90% of all asbestos currently mined and used is chrysotile. Certain studies of working populations suggested that some industrial processes might be more hazardous than others. Asbestos mining appeared to carry less of a health risk than asbestos manufacturing; and friction materials operations less than textile operations.[126] These apparent differences among fibre types and among industrial processes spurred efforts to determine whether the differences were real and, if so, what accounted for them. Controversy arose over whether the dimension of the fibre — its length and its thinness — or its chemistry explained these differences, or whether it was simply a matter of different dust levels. While these issues were being debated, fixed workplace exposure levels were being gradually reduced both as a result of improvements in equipment and technology and as a result of more stringent regulation. Occupational exposures to asbestos had to a limited extent been regulated in the past, but in light of the historical incidence of disease, it was abundantly clear that past exposure levels had been far too high. It was far less clear that the current, much lower exposure levels were still excessive. Here again, there was controversy and the latency of the asbestos-related diseases precluded an immediate answer to this controversy by direct observation.

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[122] Muriel L. Newhouse and Hilda Thompson, "Mesothelioma of Pleura and Peritoneum Following Exposure to Asbestos in the London Area," *British Journal of Industrial Medicine* 22 (1965): 261-269.

[123] See J.C. McDonald and A.D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," pp. 439-440; Acheson and Gardner, "The Ill Effects of Asbestos on Health," Table 16, p. 69 (showing 35 collected cases of mesothelioma associated with domestic exposure to asbestos); and Henry A. Anderson et al., "Household Exposure to Asbestos and Risk of Subsequent Disease," in *Dusts and Disease*, p. 155 (noting that domestic exposure cases totalled over 50).

[124] The main studies are reviewed in Chapter 5, Section B of this Report.

[125] See the discussion in Chapter 5, Section C of this Report.

[126] For a review of the major studies by industrial process, see Chapter 5, Section B of this Report.

The manifest amount of disease that had occurred in the workplace not unnaturally aroused speculation that asbestos exposures, however minimal, could cause disease in the general population. This speculation was fuelled by the incidence of mesothelioma among those not occupationally exposed and therefore thought to have inhaled only small amounts of asbestos. Moreover, opportunities for asbestos exposure among the general populace appeared to be not inconsequential. Asbestos was utilized in many consumer products; fibres were present in the water systems of most metropolitan centres; and many multi-storey schools and office buildings constructed between the 1950s and the early 1970s contained sprayed-on friable asbestos material. Friable asbestos in schools and other buildings raised another area of debate: if the material was left in place, then the occupants, whether school children or office workers, were potentially exposed; but, if the material was removed, then there were significant asbestos control problems for the maintenance and demolition workers involved. And, as all of these issues — involving both fixed and non-fixed workplaces, both occupational and non-occupational exposures to asbestos — were being debated, this Commission was appointed and went about its tasks.

Chapter 3 Asbestos in Ontario: An Overview of Health Effects and Occupational Regulation

A. The Health Effects of Past Exposures in Ontario

A.1 Introduction

Individuals may potentially be exposed to asbestos fibres in a number of ways. There are two broad categories of exposure to asbestos: occupational and non-occupational. Occupational exposure may take place in fixed workplaces, as in asbestos mining and manufacturing plants; or it may take place in non-fixed workplaces, as in construction activities or in the maintenance, repair, and demolition of buildings containing asbestos. The exposure of workers may be direct and continuous, as in the case of those employed in the manufacture of asbestos-containing products such as friction materials and textiles; or it may be incidental and intermittent, as in the case of those who, while not directly working with asbestos, may nonetheless be exposed because asbestos is present in the workplace (for example, a maintenance worker in a school with asbestos insulation).

Outside the workplace but closely related to it is the case of family members and others associated with asbestos workers who may be exposed because these workers carry asbestos on their clothing, in automobiles, etc. The general public too may be exposed to asbestos in a variety of ways. Public exposure may arise from the presence of asbestos in schools, other buildings, subways, airports, etc. It may also arise from the use of domestic products containing asbestos and from its presence as a building material in the home. In some circumstances members of the public may also be exposed to asbestos if they live in the neighbourhood of asbestos plants, whether they be mines or manufacturing facilities, or in the vicinity of waste disposal sites where asbestos may have been dumped. Finally, the public has a general environmental exposure to asbestos arising from the presence of asbestos in drinking water and in the air.

It is one thing to list the manner in which persons may be exposed to asbestos. It is quite another thing to assess how many persons have been exposed to asbestos in Ontario and the amount of such exposure. It can be said that everyone has had some exposure to asbestos in drinking water and the outdoor air. Beyond this, even among those occupationally exposed, there are no reliable data which enable us to estimate with any degree of accuracy either the number of workers exposed to asbestos in the past or the level and duration of such workplace exposure. We do know that there has been some asbestos mining in the past in Ontario, and considerably more asbestos manufacturing. We are also aware that Ontario had a shipyard industry during World War II and that, as in the rest of North America, asbestos was extensively used in the construction industry following the war.[1] However, in shipbuilding and in construction there is great uncertainty about how many employees came into contact with asbestos. In fixed place asbestos manufacturing and in mining, where the number of employees in a plant may be a proxy for the number who were actually exposed to asbestos, there is great uncertainty about the levels of exposure. This uncertainty is due in large part to the infrequency of measurements, to the fact that measurement techniques have changed, and perhaps most telling of all, to the fact that the whole method of measurement changed about fifteen years ago from the counting of dust particles to the counting of asbestos fibres.

How much disease has been caused by asbestos in Ontario? We simply do not know. There are no reliable statistics which permit us to estimate with any confidence the full extent of asbestos-related diseases resulting from past exposures. But such data as do exist chronicle a tragic legacy of disease and death resulting from workplace exposures in this province. Two sources of information in particular give us considerable cause for concern about the health effects of past exposures: the number of claims for compensation for industrial disease resulting from asbestos exposure granted by the Ontario Workers' Compensation Board (WCB); and the particular experience of the workforce employed at the Johns-Manville plant in Scarborough, Ontario.

A.2 Compensated Asbestos-Related Diseases

Allowed claims for occupational disease represent only between 1 and 2% of the over 400,000 claims allowed by the Ontario Workers' Compensation Board annually.[2] But while occupational disease claims represent only a very small proportion of total claims, they represent a considerably larger proportion of death claims. Fatal cancer provides the most striking statistic: deaths due to cancer have in recent years accounted for over 10% of all the compensated fatality claims for workers' compensation in Ontario.[3]

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[1] Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, p. 15; and The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, pp. 50–52.

Occupational cancer, including asbestos-related cancer, has been compensable by the Workers' Compensation Board since 1947.[4] Workers' awareness of the health hazards of asbestos and of their right to disease compensation grew gradually from the time of the first reports of Dr. Irving J. Selikoff's work in the mid-1960s.[5] Statistics provided to us by the Board indicate that as at the end of 1980, 111 asbestos-related cancer claims had been granted compensation: 52 for lung cancer, 48 for mesothelioma, 7 for gastrointestinal cancer, and 4 for laryngeal cancer.[6] A table published in an article, four of whose authors are Board physicians, demonstrates that as of 1978, in comparison with other occupational carcinogens, asbestos was associated with a large proportion of compensated cancer claims.[7] (These data are reproduced in Table 3.1.) Even these statistics do not tell the complete story. For while it is impossible to determine what percentage of Ontario's workers or their survivors, justifiably entitled to compensation for asbestos-related cancers, have in fact applied for benefits, it is reasonably certain that the number of claims identified and allowed by the WCB is considerably less than the total eligible for compensation.[8]

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[2] Peter S. Barth, *Workers' Compensation and Asbestos in Ontario*, Royal Commission on Asbestos Study Series, no. 2 (Toronto: Royal Commission on Asbestos, 1982), pp. 6.1–6.2. In 1980, there were 411,476 claims allowed by the WCB, of which 7,611 were industrial disease claims.

[3] Barth, *Workers' Compensation and Asbestos in Ontario*, pp. 6.1–6.2. For example, in 1980 there were 324 fatal claims allowed by the WCB, of which 42 were fatal cancer claims.

[4] Alan C. Chovil et al., "Occupational Cancer: Experience in Ontario," *Canadian Medical Association Journal* 125 (1 December 1981): 1237–1238. See also, *The Workmen's Compensation Act*, S.O. 1947, c. 119, s. 1(1)(h)(ii). Cancer in workers exposed to coal tar became compensable by amendment to the statute in 1932. See *The Workmen's Compensation Act, 1932*, S.O. 1932, c. 21, s. 10.

[5] Irving J. Selikoff, Jacob Churg, and E. Cuyler Hammond, "Asbestos Exposure and Neoplasia," *Journal of the American Medical Association* 188 (1964): 22–26. Idem, "The Occurrence of Asbestosis Among Insulation Workers in the United States," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 139–155.

[6] The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, p. 49.

[7] Chovil et al., "Occupational Cancer: Experience in Ontario," Table I, p. 1238 and text, pp. 1238–1239.

[8] See Chapter 14, Section B of this Report for a full discussion of this issue. See also, Barth, *Workers' Compensation and Asbestos in Ontario*, pp. 6.34ff.; and Paul C. Weiler, *Protecting the Worker from Disability: Challenges for the Eighties*, a report submitted to Russell H. Ramsay, Minister of Labour ([Toronto: Ontario Ministry of Labour], April 1983), p. 24.

Table 3.1

Type of Cancer and Occupational Exposure in 427 Cases in which Claims for Compensation were Accepted by the Workmen's Compensation Board of Ontario, 1947 through 1978

	Cancer Type						
Occupational Exposure	Lung	Nasal Sinuses	Mesothelioma	Gastrointestinal Tract	Larynx	Skin	Other*
Nickel refinery processes	132	43			8		
Asbestos	38		32	7	3		
Ionizing radiation, mainly from uranium	54					8	2
Coal tar and hydrocarbons	35					6	
Arsenic	24					10	
Aniline dyes							5
Other or unknown						18	2

Note: *Includes cancer of the bladder, leukemia, and melanosarcoma.

SOURCE: Alan C. Chovil et al., "Occupational Cancer: Experience in Ontario," *Canadian Medical Association Journal* 125 (1 December 1981): 1238 (Table I).

The first claim for asbestosis that was allowed by the WCB was awarded in 1942.[9] The data supplied to us by the Board indicate that to the end of 1980 the number of allowed claims for asbestosis was 222.[10] An unspecified number of these 222 claimants may have developed another asbestos-related disease.[11] When the total allowed cancer and asbestosis claims are taken together, it becomes evident that asbestos has been one of the major occupational health hazards in this province.

A.3 The Experience at the Johns-Manville Plant

Viewed in the context of the number of asbestos-related claims compensated by the WCB, the disease experience at the Johns-Manville Scarborough plant is striking. According to statistics provided to us by the Board, of the 222 allowed claims for asbestosis in Ontario as at the end of 1980, 113 came from this single plant.[12] Of the 108 allowed death claims in the province in which asbestosis or mesothelioma were involved, 51 came from the Scarborough plant.[13] The incidence of asbestosis and mesothelioma in this one plant, amounting as it does to half of all such claims compensated by the Board, has dominated the experience of the province. This dominance is even more striking when one considers that the remainder of the claims are spread widely throughout Ontario industry, with only a handful of other firms having been subject to more than one successful claim, and only two — Raybestos-Manhattan (Canada) Ltd. and Holmes Insulations Ltd. — having yielded more than 5 successful claims.[14] A Johns-Manville operation in New Toronto has yielded exactly 5 claims.[15]

One further comparison vividly demonstrates how disastrous the disease experience at the Scarborough plant has been. In response to our request, the WCB provided us with the data that establish the total number of fatal asbestos-related claims that were awarded to Johns-Manville Scarborough workers as of July 1983.[16] The number of these fatal claims is 68. This small plant, which opened in 1948 and whose total annual employment never exceeded 714 workers,[17] has occasioned more deaths from industrial disease than the entire Ontario mining industry, which annually employs over 30,000 workers, occasions from industrial accidents in an average four-year period.[18]

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[9] The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, p. 1.

[10] Ibid., pp. 9-13.

[11] Ibid. The actual data supplied by the Board list "Claims Allowed for Asbestosis, Silico-Asbestosis or Mesothelioma, 1942-1980." The total number of these allowed claims is 258. Of this figure, there are 36 allowed claims for mesothelioma without asbestosis, which by subtraction yields the number 222 for allowed claims for asbestosis. Some persons who were initially compensated for asbestosis may subsequently have developed lung, gastrointestinal, or laryngeal cancer, all of which are now compensable by the Board.

[12] The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, pp. 9-13, 20-21; and Letter from Mr. Andy Emmink, Assistant Secretary, Workers' Compensation Board to the Royal Commission on Asbestos, 29 September 1983.

[13] The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, pp. 9-13, 20-21.

[14] Ibid., pp. 9-13.

[15] Telephone communications between Mr. Andy Emmink, Assistant Secretary, Workers' Compensation Board and Royal Commission on Asbestos Staff, September 1983.

The Johns-Manville Scarborough plant has been extensively studied by Dr. Murray M. Finkelstein of the Ontario Ministry of Labour. This plant began operations in 1948. In one area of the plant, which came to be known as the Transite pipe section, asbestos-cement pipe was manufactured using a mixture of asbestos containing 4 parts chrysotile to 1 part crocidolite.[19] (A small amount of amosite was apparently used for a few years in the 1970s in an effort to improve the filtration properties of the product.)[20] In another area of the plant, rock wool insulation was manufactured using no asbestos.[21]

In 1955, the flex board shop was opened and from that year until 1970 asbestos-cement board was made in this shop using only chrysotile.[22] Then, beginning in 1960, another area of the plant was opened for the manufacture of an asbestos insulation material, called Thermobestos, using amosite asbestos. The manufacture of both asbestos-cement pipe and asbestos insulation material was discontinued in 1980.[23]

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[16] Letter from Mr. Alex Joma, Secretary, Workers' Compensation Board to the Royal Commission on Asbestos, 25 July 1983.

[17] Letter from Mr. Thomas S. Patterson, Formerly, Director, Corporate Relations, Johns-Manville Canada Inc. to the Royal Commission on Asbestos, 2 September 1983. Total annual employment in the plant reached 714 in 1975. In that year, of the 714 employees, 296 were employed in the Transite pipe section. Altogether the company estimates that approximately 5,000 workers have been employed at this plant.

[18] The average number of fatal industrial accidents which occur annually in Ontario's mining industry is approximately 16. See Canada/Ontario, *The Report of the Joint Federal-Provincial Inquiry Commission into Safety in Mines and Mining Plants in Ontario: Towards Safe Production* (Burkett Report), Kevin M. Burkett, Chairman, vol. 2: *Statistics and Research Reports* (Toronto: April 1981), p. 15.

[19] Murray M. Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," *British Journal of Industrial Medicine* 40 (1983): 138–144. Dr. Finkelstein, in his testimony before us, indicated that the percentage of crocidolite in the final product was approximately 3%. Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 19. Information was also provided to the Royal Commission on Asbestos by Johns-Manville Canada Inc., 1982.

[20] RCA Transcript, Evidence of Mr. Bruce C. Machin, 28 June 1982, Volume no. 45(A), p. 116; Ontario, Royal Commission on Asbestos, Exhibit II-68 [hereafter RCA Exhibit], in RCA Transcript, Evidence of Mr. Bruce C. Machin, 28 June 1982, Volume no. 45(A), p. 62: Manville, Internal Correspondence from Mr. R. Dennis Stevens, Toronto Plant to Mr. Bruce C. Machin, West Mall, 23 June 1982.

[21] Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 138.

[22] Ibid. See also, RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 19–21.

During the first twelve years of its operation, such dust control as existed in the Transite pipe section of the plant was provided by systems installed at the time of its construction.[24] Further attempts to control dust in this part of the plant led to two major ventilation changes. The first, the installation of a new dry-end dust collector system, involved a 1961 capital expenditure of $139,000 and, according to Dr. Finkelstein, came into operation in 1962. The second, the installation of a new wet-end dust collector and a new fibre handling collector, involved a 1968 capital expenditure of $640,542 and, again according to Dr. Finkelstein, came into operation in 1970.[25] A double locker system for the employees in the Transite pipe section and new showers were installed in 1976 at an expenditure of $170,000.[26] The Transite pipe section was closed in 1980.

The morbidity and mortality experience of the workers at the Johns-Manville Scarborough plant have been the subject of two recently published studies by Dr. Finkelstein,[27] who gave testimony before this Commission at its formal hearings in the summer of 1981.[28] His articles, published in 1982 and 1983, and his subsequent work have enlarged upon his oral evidence.

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[23] Ibid.

[24] RCA Exhibit II-67(a), in RCA Transcript, Evidence of Mr. Bruce C. Machin, 28 June 1982, Volume no. 45(A): Johns-Manville, Internal Correspondence from Mr. K.H. Reeve, Toronto Plant to Mr. J.R. Ariss, CPDHQ, Etobicoke, 24 December 1980.

[25] Ibid. In his publications, Dr. Finkelstein has indicated that these major ventilation changes were actually effected in 1962 and 1970. See Murray M. Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," *American Review of Respiratory Disease* 125 (1982): 497. In the unpublished version of this article, Dr. Finkelstein stated that:

> Two major ventilation changes were made in the pipe plant over the years of operation. In 1962, the main duct system in the finishing end of the plant was replaced with new fans, and a baghouse was installed. The ventilation capacity was increased from 45,000 to 60,000 cfm. The old finishing end equipment was serviced for use in the forming end, and ventilation capacity there was subsequently increased from 35,000 to 45,000 cfm. The second major change occurred in 1970 when a negative pressure system of 55,000 cfm capacity was installed in the forming end. [Murray M. Finkelstein, "Asbestosis Among Long-Term Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1981, p. 9. (Mimeographed.)]

See also, RCA Transcript, Evidence of Mr. Bruce C. Machin, 28 June 1982, Volume no. 45(A), pp. 54–57.

[26] RCA Exhibit II-67(a), Johns-Manville, Internal Correspondence from Mr. K.H. Reeve to Mr. J.R. Ariss, p. 6.

[27] Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," pp. 496–501. Idem, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," pp. 138–144.

[28] Dr. Finkelstein testified on 28 July 1981 and again on 26 August 1981. See RCA Transcript, Volume no. 24 and Volume no. 30 respectively.

Dr. Finkelstein first studied only those workers who were hired prior to 1960 and were employed by the company for at least 9 years.[29] His cohort thus constituted 339 men, of whom 252 had at least one year's exposure to asbestos (either in the pipe shop, the cement board shop, or in maintenance work), while the remaining 87 worked in the rock wool insulation operation or were otherwise minimally exposed to asbestos. Dr. Finkelstein traced the mortality experience of these workers to the end of 1980. The employees in rock wool insulation had mortality rates similar to those of the general Ontario population, while the group of asbestos-exposed employees had an overall mortality rate double the general Ontario population. Overall cancer mortality rates among the asbestos workers were 5 times greater than would be expected in the general population, and deaths attributable to lung cancer alone were 8 times more frequent than would be expected in the general population.[30] Of the 58 deaths among the 186 production workers exposed to asbestos in the manufacture of asbestos-cement pipe or asbestos-cement board, over half, 30, were either from lung cancer (20 deaths) or mesothelioma (10 deaths);[31] and in the total workforce studied by Dr. Finkelstein, there were no fewer than 16 mesothelioma deaths, only one of whom had reached retirement age by the time of his death.[32] One of our expert witnesses, Dr. Philip E. Enterline of the University of Pittsburgh, testified before this Commission as to the mortality experience of these workers in the following sobering words: "I've seen those data. They are remarkable data. . . . I don't know of anything quite like that in the literature."[33]

Dr. Finkelstein has attempted to estimate the cumulative exposure of the workers in his cohort on the basis of the available measurement data and communications with company industrial engineers, and having regard to the Transite pipe section's major ventilation changes made in 1962 and 1970.[34] Measurement data from the plant were meagre. There were annual dust counts by government or insurance company hygienists in only five of the years between 1948 and 1961. In 1961, Johns-Manville itself began annual dust measurements with an instrument known as the midget impinger.

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[29] Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 139.

[30] Ibid., p. 140.

[31] Ibid., pp. 140–141. These figures are based upon "best evidence." In other words, in addition to the official death certificate codings, the author had further information from clinical, pathological, and necropsy reports.

[32] RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 79–80; and 26 August 1981, Volume no. 30, p. 8. Again, this number is based on "best evidence." In the case of mesothelioma, rather than accepting the cause of death as stated on the death certificate, the author, due to the difficulty of diagnosing mesothelioma, had a diagnosis made either by the Canadian Tumour Reference Centre or by Dr. Alexander C. Ritchie, Professor of Pathology at the University of Toronto.

[33] RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, p. 141.

[34] Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 497; and RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 52–53, 127–129.

Fibre counts on at least a quarterly basis were made by the company, beginning in 1969, utilizing the membrane filter method.[35] Dr. Finkelstein attempted to reconstruct historical exposure levels on the basis of the company's membrane filter counts taken immediately prior to the 1970 ventilation change. He discussed historical conditions with two industrial engineers from Johns-Manville (one who was present when the plant was being built and worked there until 1969, and the other who worked in the plant in the 1950s and designed the 1970 ventilation changes).[36] On the basis of these discussions and of fragmentary historical dust counts, he assumed that exposures between 1948 and 1954 were twice as high as those immediately prior to the 1970 ventilation change, that exposures between 1955 and 1962 were 30% higher, and that exposures after 1962 were at the levels recorded prior to the 1970 change. These assumed exposure levels, together with the recorded asbestos-related disease, permitted Dr. Finkelstein to correlate asbestos exposure levels with disease.[37] He found that, at an estimated cumulative exposure of 100 fibres per cubic centimetre-years (f/cc-yrs), lung cancer mortality rates were elevated over eightfold among the asbestos-exposed workers.[38]

The exposure estimates made by Dr. Finkelstein have been questioned as being too low, and the author himself has fairly admitted they are fraught with uncertainty.[39] One source of uncertainty involves work practices at the plant. Based on the testimony we heard, work practices which were quite indifferent to the hazardous nature of asbestos were common and likely gave rise to sporadic exposures of a very high level. The current

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[35] Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 497; and RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 127. See also, Chapter 5, Section B of this Report.

[36] RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 52-53; and RCA Transcript, Evidence of Mr. Edgar G. Stevens, 10 August 1982, Volume no. 54, p. 50.

[37] Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 497; and RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 52-53. Dr. Finkelstein testified that the factor of 2 increase to estimate exposures for the period 1948-1954 was based on his discussions with company personnel and on the generally held view that dust conditions in the plant were "pretty bad" in that period.

[38] Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 143.

[39] RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 56-59. See also, RCA Transcript, Evidence of Mr. Julian Peto, 30 July 1981, Volume no. 25(B), p. 5, where Mr. Peto suggested that it is possible that Dr. Finkelstein's estimates of fibre counts were underestimates, and that there may have been much higher crocidolite levels than his estimates would suggest. See also, letter from Dr. Hans Weill, Pulmonary Diseases Section, Department of Medicine, Tulane Medical Center, New Orleans, Louisiana to the Royal Commission on Asbestos, 21 July 1982, observing the striking uncertainty of the Finkelstein exposure estimates.

Vice-President of Manville Canada Inc. recalled his experience as an employee at the Scarborough plant in 1963 in the following words:

> I can recall one of my initial making the rounds with the chap who was union president for many years, that I found out how to work a dust collector by putting my head inside the hopper bin and having somebody turn on the shaker and bury me in the hopper, with asbestos. That would be in 1963, when I was in maintenance . . . which was thought to be quite humorous at the time.[40]

A former employee, Mr. Nick Carrigan, a member of the Asbestos Victims of Ontario, spoke of historical conditions in these terms:

> In the old days, we had a wheelabrator system where it worked on a timer. At certain times the dust system was shut down, at break periods in particular, and we had what we call . . . we put a vibrator . . . we pressed a button, a vibrator went on the bins to shake the bags. You wouldn't believe . . . you couldn't see yourself. You couldn't see the next guy sitting beside you. And while this was going on, we sat down and we ate our lunch in it.
>
> Now we also willowed blue asbestos fibre, mined in South Africa, which is supposed to be the most deadly of them all. We willowed that raw fibre into what we called a spare bin, and then we handled it. We pulled it out of there without masks, without protective clothing, we pulled that blue asbestos fibre out into buckets and dumped it into the back of the machine.
>
> We had leaks, unbelievable asbestos leaks, all over the place, never fixed. That only came in later years when the pressure was put on a little.[41]

Still, the company's own personnel to whom Dr. Finkelstein submitted his exposure estimates thought they were not unreasonable, and Dr. Finkelstein himself suggested that they are at least accurate to within a factor of 3 to 5.[42] It should be noted that the underlying data on which these estimates were based are no more uncertain than the data available for most historical studies of asbestos-exposed workers.[43] As much as anything else, the uncertainty of Dr. Finkelstein's exposure estimates illustrates how little we really know about past levels to which asbestos workers were subjected.

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[40] RCA Transcript, Evidence of Mr. Bruce C. Machin, 28 June 1982, Volume no. 45(A), p. 111.

[41] RCA Transcript, Submission by Mr. Nick Carrigan, Asbestos Victims of Ontario, 16 February 1981, Volume no. 1, p. 180.

[42] Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 143; Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," pp. 497–498; and RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 56–64.

Dr. Finkelstein has now extended his mortality study to include all workers hired before 1960 who were employed one year or more — a total of 535 asbestos-exposed and 205 non-exposed employees — and followed them through 1981.[44] Despite the brevity of the employment criterion for entry to the cohort, the disease incidence remains extraordinarily high. The overall cancer mortality rate is nearly 4 times greater than that of the general population, the lung cancer rate alone nearly 5 times greater, and the rate for gastrointestinal cancer nearly 3 times greater.[45] A comparison of the results of Dr. Finkelstein's study with published studies of other asbestos workers such as those at Rochdale, England, and Charleston, South Carolina, forces us, regretfully, to conclude that the disease experience of the workforce at this plant is in line with the worst asbestos-related health experiences observed anywhere in the world.[46] Indeed, it constitutes nothing less than a world-class occupational health disaster.

This world-class occupational health disaster has not yet run its course. There is a distinct possibility, indeed probability, that more asbestos-related deaths are yet to come among the workers who were employed at this plant. Asbestos-caused diseases do not generally manifest themselves until at least 10 to 15 years after initial exposure and more often occur more than 20 years from the time of first exposure to asbestos. The mortality experience of the Johns-Manville workers reported by Dr. Finkelstein is the result of asbestos exposures many years ago. Dust levels were indeed reduced in this plant over time, and this we expect will have some effect in reducing the incidence of disease.[47] But we must recognize the possibility that as time goes on, the toll of deaths from the past inhalation of asbestos will continue to mount. Telling evidence that this has been happening is found in the continuing work of Dr. Finkelstein who, by August 1982, had observed 21 deaths from mesothelioma among the study population at the plant, an increase of 5 in the 12 months that had passed from the time he last testified before the Commission.[48] All but one of the 21 victims were exposed both to chrysotile and crocidolite asbestos in the pipe plant or in maintenance work.[49] At this time, we can only speculate as to what the future holds for the other plant employees, but we cannot fail to observe that at most they are at a distance of but 35 years from first exposure to asbestos at this plant, and that the employees hired in 1960 or later, who have yet to be studied by Dr. Finkelstein, have barely reached, if at all, 20 years from first exposure.

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[43] See the discussion of the uncertainty of historical exposure data in Chapter 4, Section D of this Report. See also, the review of the major studies in Chapter 5, Section B of this Report.

[44] Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983. (Mimeographed.)

[45] Ibid., Table 2.

[46] See, for example, John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," *Annals of Occupational Hygiene* 26:1–4 (1982): 869–887; Julian Peto, "The Hygiene Standard for Chrysotile Asbestos," *The Lancet* 1 (4 March 1978): 484–489; Julian Peto, "The Incidence of Pleural Mesothelioma in Chrysotile Asbestos Textile Workers," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 703–711; and Julian Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 829–836. See also, RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 123.

B. History of Occupational Regulation of Asbestos in Ontario

Occupational exposure to asbestos in Ontario has been regulated by a combination of: (i) legislative provisions generally directed to the health of workers in the mining, construction, and industrial sectors of the economy; (ii) legislative provisions specific to asbestos; and (iii) non-legislative guidelines directly applicable to asbestos exposure and use.[50] As we shall discern, from the 1940s onwards regulatory practices in the United States and the United Kingdom had a considerable influence in Ontario.

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[47] Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 497; and Johns-Manville Canada Inc., Written submission to the Royal Commission on Asbestos, #18, 15 January 1981, Appendix A.

[48] Murray M. Finkelstein, "Mortality in Asbestos-Cement Factory Workers," paper presented at the Second International Symposium on Epidemiology in Occupational Health, Montreal, 25 August 1982, p. 4. When Dr. Finkelstein first testified before this Commission in July 1981, he gave evidence that there were in total 15 mesothelioma deaths of which he was aware among the Johns-Manville workers. RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 79–80. When Dr. Finkelstein returned to complete his testimony in August 1981, he had discovered another mesothelioma, bringing the total to 16. RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 26 August 1981, Volume no. 30, pp. 2–8.

[49] Finkelstein, "Mortality in Asbestos-Cement Factory Workers," pp. 4–5. Dr. Finkelstein, in his September 1983 article, noted:

> There were 21 deaths from mesothelioma in the cohort; 19 have had pathological review. Seventeen of these deaths occurred among the production workers with 2 each among the maintenance and control groups. One of the control group spent the first part of his employment in Rock Wool/Fibreglass and was transferred into the Pipe shop after the 1961 classification date; the other man is not recorded as having worked in the asbestos areas. It was possible, however, for men to be assigned to the Pipe shop for brief cleanup duties and he may have been exposed in this fashion. (Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," pp. 9–10.)

Heavy concentrations of dust in the workplace were recognized as a health hazard long before asbestos was widely used. For example, *The Ontario Factories Act* of 1884 provided that all ventilation in factories must ". . . render harmless, so far as is reasonably practicable, all the gases, vapours, dust or other impurities generated. . . ."[51] An amendment to *The Mining Act* in 1912 provided that dust be removed where present in the air in a quantity "injurious to health"[52] and a similar amendment was added to *The Factory, Shop and Office Building Act* in 1913.[53] This general legislative framework in both the industrial and mining sectors remained in place with minor amendments until the 1960s and was available to control asbestos exposures once asbestos was identified as a serious occupational hazard. However, within this legislative framework there were no specific exposure limits for asbestos. Equally important, the legislation which regulated the construction industry where asbestos was widely used, *The Buildings Trades Protection Act*, 1911, focused almost exclusively on safety and made no reference to health hazards whatsoever.[54]

In 1946, the American Conference of Governmental Industrial Hygienists (ACGIH), a professional organization in the United States, voluntarily assumed the task of formulating recommended occupational health standards, a task which was to influence occupational health policy in Ontario for the next quarter of a century. Acting on the recommendations of Dreessen et al. in 1938 following a study of 541 workers in four asbestos textile plants, the ACGIH adopted a workplace exposure limit, for which it registered the trademark name Threshold Limit Value (TLV). The TLV for asbestos was 5 million particles per cubic foot (mppcf) and was a time-weighted average concentration based on impinger sampling and counted by optical microscopy.[55] The asbestos TLV of 5 mppcf remained operative as a guideline in the United States until 1970. In 1947, the Occupational Health Branch of the Ontario Ministry of Health began using this TLV of 5 mppcf as a criterion in issuing control orders and in making recommendations to employers following air sampling.[56] As in the United States, this TLV was not in and of itself a legally enforceable standard. Rather, it served as a guide to the proper control of dust in the workplace.[57] Moreover, it dictated a count of all dust particles present in the workplace even while serving as a guideline for the control of asbestos where present. The TLV of 5 mppcf remained the recommended standard for fixed place industry in this province for 20 years,[58] thus spanning most of the period for which the Johns-Manville workers studied by Dr. Finkelstein were exposed.

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[50] For a general history of occupational regulation of asbestos, see Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, pp. 10-25; and Sandra Glasbeek, *A Survey of Asbestos Policies in Canada with Particular Emphasis on Ontario*, Royal Commission on Asbestos Background Paper Series, no. 1 (Toronto: Royal Commission on Asbestos, 1981).

[51] *The Ontario Factories Act*, 1884, 47 Vict., c. 39, s. 11(3).

[52] *The Mining Amendment Act 1912*, 2 Geo. V, c. 8, s. 18, par. 59.

[53] *The Factory, Shop and Office Building Act*, 1913, 3-4 Geo. V, c. 60, s. 43(13).

[54] *The Buildings Trades Protection Act*, 1911, 1 Geo. V, c. 71.

Meanwhile, in 1962, *The Construction Safety Act* came into force and gave recognition for the first time to health hazards in the construction industry.[59] A regulation made under that Act provided that no worker was to be present where there was a hazard of injury from inhaling a noxious gas, fume, or dust unless he was protected by mechanical ventilation or the wearing of a suitable respirator.[60] A further regulation under that Act made in 1973 established conditions under which asbestos sprays could be applied which were so stringent that they quickly led to their elimination.[61]

As for fixed place industry, *The Factory, Shop and Office Building Act* was repealed in 1964 and replaced by *The Industrial Safety Act, 1964*.[62] This new statute gave the Minister of Labour far more extensive powers to deal with exposures to toxic substances in industry. A regulation passed under this Act made specific reference to asbestos as one of the air contaminants which must be exhausted from the workplace in such a manner as to prevent its return.[63] The Act and regulations were revised in 1971-1972, broadening the control over fixed workplace exposures to asbestos.[64]

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[55] Waldemar C. Dreessen et al., *A Study of Asbestosis in the Asbestos Textile Industry*, U.S. Public Health Bulletin, no. 241 (Washington, D.C.: U.S. Government Printing Office, August 1938): 68–69. This study has been severely criticized because of its apparent flaws. Only 13% of the group surveyed had been employed 10 or more years and only 3 workers had been employed more than 20 years. Further, prior to the study the plants apparently discharged 150 workers suspected of having asbestosis. See William J. Nicholson, "Regulatory Actions and Experiences in Controlling Exposure to Asbestos in the United States," *Annals of the New York Academy of Sciences* 329 (26 October 1979): 293–299.

[56] Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, p. 17.

[57] In other words, there was no specific statutory provision which created an offence for exceeding the control limit. However, the government could prosecute an employer for failure to control dust and use as evidence the fact that the Threshold Limit Value had been exceeded. This illustrates the difference between a legally enforceable standard and a guideline.

[58] Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, pp. 17–18.

[59] S.O. 1961–1962, c. 18.

[60] O. Reg. 269/69, s. 31, revised under *The Construction Safety Act, 1961–62*.

[61] O. Reg. 419/73.

[62] S.O. 1964, c. 45.

In the late 1960s, as the technology of measurement improved, the method of measuring workplace exposure to asbestos changed from a count of dust particles to a count of fibres. For measurement purposes an asbestos fibre was defined in accordance with common international practice as a fibre longer than 5 microns[65] whose length to diameter ratio (aspect ratio) was at least 3 to 1. This definition originated in the United Kingdom, having been somewhat arbitrarily selected by three asbestos manufacturers who collaborated on the matter. It was apparently chosen to facilitate counting on the standard instrument of measurement, the optical microscope, and because it was thought that the development of asbestosis was related to longer fibres.[66]

In 1968, the ACGIH referred for the first time to fibre counts as well as particle counts and published a new intended TLV for asbestos of 2 mppcf or 12 fibres per cubic centimetre (f/cc). Two years later, in 1970, the ACGIH, now referring to fibre counts alone, proposed a new TLV of 5 f/cc applicable to all types of asbestos and officially adopted this value in 1973.[67] The Ontario Ministry of Health accepted the proposed values published by the ACGIH in making assessments of asbestos exposures in Ontario industry. The Ministry of Labour, utilizing the findings of the Ministry of Health, continued to issue orders on the basis of these assessments.[68]

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[63] O. Reg. 196/64, s. 20(c), under *The Industrial Safety Act, 1964*.

[64] *The Industrial Safety Act, 1971*, S.O. 1971, c. 43; and O. Reg. 259/72.

[65] The correct term for one-millionth of a metre is micrometre, but we will use the more familiar term micron.

[66] This definition was also adopted in light of the evidence then available which suggested that the particle size distribution of an asbestos dust cloud was reasonably constant over a wide range of textile processes. Later work indicated this might not be true. See S. Holmes, "Developments in Dust Sampling and Counting Techniques in the Asbestos Industry," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 289; and C.G. Addingley, "Asbestos Dust and Its Measurements," *Annals of Occupational Hygiene* 9 (April 1966): 73-82. The three asbestos manufacturers were Turner Brothers Asbestos Co. Ltd. (Rochdale), British Belting and Asbestos Ltd. (Yorkshire), and Cape Asbestos Co. Ltd. (Barking), who together formed the Asbestosis Research Council to consider problems of dust sampling as well as the medical aspects of asbestosis. See also, RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 47; RCA Transcript, Evidence of Dr. Graham W. Gibbs, 13 November 1981, Volume no. 33, pp. 29-30; Eric J. Chatfield, "Measurement of Asbestos Fibres in the Workplace and in the General Environment," in Mineralogical Association of Canada, Université Laval, Québec: 20-22 May 1979, *Short Course in Mineralogical Techniques of Asbestos Determination*, ed. R.L. Ledoux (Toronto: Mineralogical Association of Canada, Royal Ontario Museum, May 1979), p. 112; Murray M. Finkelstein, "On an Occupational Standard for Exposure to Asbestos," Appendix 9 to Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, Appendices, p. 74.

[67] Irving J. Selikoff and Douglas H.K. Lee, *Asbestos and Disease* (New York: Academic Press, 1978), pp. 443ff.; and Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, pp. 18-19.

But while the ACGIH was reformulating its TLVs in the late 1960s, a study in the United Kingdom was published which was to have a significant effect on the regulation of asbestos in North America. Asbestos had been regulated in the United Kingdom since 1931 by the Asbestos Industry Regulations passed under the "Dangerous and Unhealthy Industries" section of the *Factory and Workshop Act* of 1901.[69] These regulations contained extensive provisions for dust control but not exposure limits. In 1968, the Committee on Hygiene Standards of the British Occupational Hygiene Society (BOHS) recommended that occupational exposures to chrysotile asbestos be limited to 2 f/cc and occupational exposures to crocidolite be reduced to 0.2 f/cc.[70] The BOHS recommendation for chrysotile was predicated on reducing asbestos exposures to the point where the risk of contracting early signs of asbestosis would be only 1% after a lifetime exposure of 50 years. It was based upon a review of available literature and, in particular, upon a BOHS study of employees in the British asbestos textile mill at Rochdale.[71] The BOHS workplace recommendation of 2 f/cc was subsequently subjected to considerable criticism, but nevertheless it soon served as a standard for many other jurisdictions, among them the United States and Ontario.[72] In the United States, the 2 f/cc standard was recommended by the National Institute for Occupational Safety and Health (NIOSH) in 1972 and adopted by the Occupational Safety and Health Administration (OSHA) as the official standard in that country in 1976.[73] Despite subsequent proposals to lower the limit, this remains the standard in effect in the United States, although OSHA promulgated in November of 1983 an Emergency Temporary Standard of 0.5 f/cc, which was subsequently stayed by court order.[74]

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[68] Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, pp. 17-19.

[69] U.K., Asbestos Industry Regulations (1931), Statutory Rules and Orders, 1931, no. 1140 (London: His Majesty's Stationery Office, 1931), under the *Factory and Workshop Act*, 1901.

[70] British Occupational Hygiene Society, Committee on Hygiene Standards, "Hygiene Standards for Chrysotile Asbestos Dust," *Annals of Occupational Hygiene* 11 (1968): 47-48.

[71] Ibid., Appendix (i), pp. 54-58.

[72] See particularly, Peto, "The Hygiene Standard for Chrysotile Asbestos"; Geoffrey Berry et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory," *British Journal of Industrial Medicine* 36 (May 1979): 98-112; RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 54; RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 49; and RCA Transcript, Evidence of Mr. Julian Peto, 30 July 1981, Volume no. 25(B), pp. 25-28. The British Occupational Hygiene Society's 2 f/cc standard has been criticized, first because it was a standard based on the prevention of asbestosis only and not cancer, and second because it failed to take adequate account of the fact that asbestosis is a progressive disease even in the absence of further exposure. In fact, the population at Rochdale, whose health experience was studied, was an employed population. Accordingly, no account was taken of persons who had been exposed to asbestos but who had either left Rochdale or who had been moved to other areas in the plant prior to the study having been undertaken. Accordingly, persons who had to terminate their exposure to asbestos or their employment altogether because of illness and those persons who developed symptoms of asbestosis after their employment had ceased were not properly considered in the study.

Ontario acted on the BOHS recommendation three years earlier than the United States. In 1973, the Ministry of Health abandoned the TLVs of the ACGIH as a reference point for asbestos and adopted as its guideline the time-weighted average exposure limit of 2 f/cc for all types of asbestos.[75] This guideline became effective not only in the industrial sector but in the mining sector as well. Then, in 1975, the Ministry of Health, through its Occupational Health Branch, adopted a guideline of 0.2 f/cc for the amphiboles.[76] For the first time, the Ontario government had adopted a guideline which differentiated exposure limits on the basis of fibre type.

In 1978, following the recommendations of the Royal Commission on the Health and Safety of Workers in Mines, all provincial health and safety legislation in Ontario was consolidated under *The Occupational Health and Safety Act, 1978*, with administrative responsibility vested in the Ministry of Labour.[77] The Act was proclaimed into force on October 1, 1979.[78]

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[73] U.S., National Institute for Occupational Safety and Health, *Revised Recommended Asbestos Standard*, prepared by Richard A. Lemen and John M. Dement (Washington, D.C.: U.S. Department of Health, Education and Welfare, December 1976). For a history of U.S. regulatory action, see Nicholson, "Regulatory Actions and Experiences in Controlling Exposure to Asbestos in the United States," pp. 294–297.

[74] RCA Exhibit II-3, in RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8: Philip E. Enterline, "Epidemiologic Basis for the Asbestos Standard," paper presented at the Second Annual Symposium on Environmental Epidemiology, Pittsburgh, Pennsylvania, 28 April 1981. (Mimeographed.) See also, U.S., Department of Labor, Occupational Safety and Health Administration, "Occupational Exposure to Asbestos; Emergency Temporary Standard," 29 CFR Part 1910, 48 FR 51086–51140, 4 November 1983.

[75] Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, p. 19.

[76] Ibid.

[77] S.O. 1978, c. 83.

[78] S.O. 1979, Table of Proclamations, p. 858. Three regulations were passed pursuant to this statute: the Regulation for Industrial Establishments, O. Reg. 658/79 as amended; the Regulation for Construction Projects, O. Reg. 659/79 as amended; and the Regulation for Mines and Mining Plants, O. Reg. 660/79 as amended. (See now, R.R.O. 1980, Regulations 692, 691, and 694 respectively.) Only the Regulation for Mines and Mining Plants contained an enforceable control limit for exposure to asbestos, incorporating by reference for underground mines only the 1979 TLVs of the ACGIH. For asbestos, the TLVs prescribed exposure standards of 0.2 f/cc for crocidolite, 0.5 f/cc for amosite and tremolite, and 2.0 f/cc for chrysotile and all other forms of asbestos.

However, the Ontario government had publicly declared its intention to regulate general occupational health hazards and the exposure of employees to asbestos in particular, under legally enforceable control limits, even before the new legislation was proclaimed.[79] Once in force, section 41 of *The Occupational Health and Safety Act* gave the Lieutenant Governor in Council power to make regulations designating any substance and, once designated, prohibiting, regulating, or limiting the handling, exposure, use, and disposal of such a substance. On June 28, 1980, shortly after the appointment of this Commission, the Ministry of Labour published in *The Ontario Gazette* notice of intent to designate asbestos, together with five other substances and noise.[80] On August 16, 1980, a proposed asbestos regulation was published, together with a request for public comment from workers, employers, and other interested parties.[81] In setting exposure limits under the proposed regulation, the Ontario government again drew upon British advice. In 1979, the United Kingdom Advisory Committee on Asbestos had tabled its final report.[82] Among its recommendations were that the time-weighted average control limits for exposure to crocidolite, amosite, and chrysotile asbestos be 0.2, 0.5, and 1.0 f/cc (over a 4-hour sampling period) respectively.[83] This reflected the view of the Advisory Committee that different fibre types were of different relative pathogenicity; and that crocidolite was the most pathogenic, chrysotile the least, with amosite occupying an intermediate position. The asbestos regulation proposed by the Ontario Ministry of Labour in August of 1980 adopted this approach and these numbers (but over a 40-hour averaging period) and also provided that the maximum allowable concentration in any period of time be 2.0 f/cc for crocidolite and amosite and 5.0 f/cc for chrysotile and all other types of fibres.[84] The proposed regulation on asbestos was revised and ultimately was filed and became effective in law in

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[79] Ontario, Ministry of Labour, "Notice of Intent to Regulate Lead, Asbestos and Silica and Occupational Health Hazards," *The Ontario Gazette*, vol. 111-29, 22 July 1978, p. 3546.

[80] Ontario, Ministry of Labour, "Notice of Intention," *The Ontario Gazette*, vol. 113-26, 28 June 1980, p. 2641.

[81] Ontario, Ministry of Labour, "Proposed Regulation under *The Occupational Health and Safety Act, 1978:* Asbestos — Designated Substance," *The Ontario Gazette*, vol. 113-33, 16 August 1980, pp. 3339-3348.

[82] U.K., Advisory Committee on Asbestos, *Asbestos — Volume 1: Final Report of the Advisory Committee* (Simpson Report), William J. Simpson, Chairman (London: Her Majesty's Stationery Office, 1979).

[83] Ibid., Recommendation 14, p. 74, regarding crocidolite; Recommendation 17, p. 78, regarding amosite; and Recommendation 15, p. 77, regarding chrysotile. In fact, the Advisory Committee recommended that there should be a statutory ban on the import of raw crocidolite fibre and that the control limit for crocidolite already in place be 0.2 f/cc.

[84] Ontario, Ministry of Labour, "Proposed Regulation under *The Occupational Health and Safety Act, 1978:* Asbestos — Designated Substance," *The Ontario Gazette*, vol. 113-33, 16 August 1980, s. 4(2) and Schedule, pp. 3340, 3348.

August 1982, more than two years after the notice of intent was published.[85] Under the Regulation, asbestos was prescribed as a designated substance; instead of being simply a guide to proper dust control, the numerical exposure limits became legally enforceable standards; these exposure limits were unchanged from the August 1980 proposal, save for the maximum allowable concentration for crocidolite, which was reduced from 2.0 f/cc to 1.0 f/cc, and for amosite, which was raised from 2.0 f/cc to 2.5 f/cc. And significantly, the employer was placed under an explicit obligation not merely to ensure that the exposure limits were not exceeded but also to ensure that the time-weighted average exposure of workers was reduced to the lowest practical level.[86] The Regulation Respecting Asbestos was made applicable to the industrial and mining sectors and became, at the time of its promulgation, one of the most stringent of any jurisdiction in the world, Sweden being a notable exception.[87]

Asbestos on construction projects is to be subject to a separate regulation. In October 1981, the Ministry of Labour circulated guidelines for the removal and treatment of asbestos on construction projects and published, in August 1982, a Proposed Regulation Respecting Asbestos on Construction Projects,[88] followed by a revised proposal in January 1983.[89] The proposed regulation reflects the difficulties inherent in monitoring exposures to hazardous substances in non-fixed site workplaces. Rather than prescribing exposure limits for the control of asbestos, it sets out a detailed list of work practices to be followed — in other words, "regulation by procedure." As of the summer of 1983, the Proposed Regulation Respecting Asbestos on Construction Projects remained under consideration within the Ministry of Labour.

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[85] Regulation Respecting Asbestos, O. Reg. 570/82, made under the *Occupational Health and Safety Act,* R.S.O. 1980, c. 321. The mining regulation has not been changed so that in theory the TLVs of the ACGIH still apply to underground mines. But we are advised that it is the intent of the Ontario government to make the Regulation Respecting Asbestos applicable to all mining and to amend ss. 243 and 244 of the Regulation for Mines and Mining Plants so that s. 279 will be inapplicable in the case of a designated substance.

[86] Regulation Respecting Asbestos, ss. 2 and 4.

[87] Arne Westlin, "The Swedish Experience in Banning and Restricting Certain Applications of Asbestos and in the Use of Substitutes," paper presented at *Engineering Occupational Health,* a symposium sponsored by the Department of Chemical Engineering and Applied Chemistry, University of Toronto, Canadian Society for Chemical Engineering (Toronto Section), Chemical Institute of Canada (Toronto Section), and Ontario Ministry of Labour, Toronto, 13 April 1978. (Mimeographed.) In a copy of a telex to the British Embassy in Washington and dated 24 August 1983, sent by Mr. Cyril D. Burgess of the U.K. Health and Safety Executive to the Royal Commission on Asbestos, it was stated, *inter alia,* that the Health and Safety Commission had decided that: "1. Exposure limits for chrysotile to be reduced to 0.5 fibres per ml and for amosite to 0.2 fibres per ml effective from 1 August 1984. 2. A ban should be placed on the importation and use in manufacture of amosite and products containing it. Prohibition regulations should be brought forward for amosite and crocidolite to come into effect on 1 June 1984. . . ."

[88] Ontario, Ministry of Labour, Occupational Health and Safety Division, "Notice of Proposed Regulation: Designated Substance — Asbestos on Construction Projects," *The Ontario Gazette,* vol. 115-33, Saturday, 14 August 1982, pp. 3194–3197.

[89] Ontario, Ministry of Labour, Occupational Health and Safety Division, "Proposed Regulation Respecting Asbestos on Construction Projects and Related Codes," presented at a Public Meeting, 17 January 1983, Toronto. (Mimeographed.)

From this brief history, it can be seen that the Ontario government, in evolving its regulation of occupational exposure to asbestos, has borrowed freely and frequently from practices in the United States and in the United Kingdom. But the history of Ontario's experience is not only one of sharing U.S. and U.K. practices. It is one that reflects the slow recognition in all countries of workplace environmental factors as determinants of health.

Chapter 4 Sources of Information on the Health Effects of Asbestos

A. Introduction

In his address called "A Review of Diagnostic Tests for Causation" at the Second Public Meeting of this Commission held on December 12, 1980, Dr. David L. Sackett, Professor of Clinical Epidemiology and Biostatistics and Professor of Medicine at McMaster University, pointed out that two different kinds of expertise are required in order to understand the health effects of asbestos exposure.[1] First, there is a need for methodological expertise in the critical assessment of information concerning the etiology, that is, the causation of asbestos-related diseases. Second, there is a need for biological expertise in the subject matter of asbestos and its effects on the cells, tissues, and organs of the body.

During the course of our inquiry we heard sworn testimony from a substantial number of the world's acknowledged experts on the health effects of asbestos. These witnesses imparted to us their expertise on the two subjects to which Dr. Sackett referred in his public address. In addition, we have digested a good deal of the voluminous and proliferating medical literature on asbestos. In the next two chapters we report on what we have learned.

In this chapter we first consider the biological evidence, what we know and what we do not know, about the biological mechanisms by which asbestos causes disease. We next discuss in general terms the principal sources of information on the health effects of asbestos and the criteria by which that information ought to be assessed. Then we offer our judgement as to the strengths, weaknesses, and limitations of the available evidence. Our purpose is to provide a foundation which will enable us to address, both in Chapter 5 and in the remainder of this Report, the health issues that are of current and future concern.

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[1] David L. Sackett, "A Review of Diagnostic Tests for Causation," in Ontario, Royal Commission on Asbestos, *Proceedings of The Royal Commission on Asbestos, Second Public Meeting, Friday, December 12, 1980*, reported by Lydia Dotto (Toronto: Royal Commission on Asbestos, 1981), p. 10 and Appendix B, p. 1.

B. The Biological Evidence

We turn first to the biological evidence pertaining to the pathogenesis of asbestos-related diseases. We do so with considerable trepidation. Current understanding of the biological mechanisms which induce asbestos-related cancer or indeed asbestosis is limited. Much remains in the realm of speculation and hypothesis. Our justification for raising the subject is the prospect that what we do know about these mechanisms will assist us to determine the biologically relevant characteristics of asbestos fibres and thereby permit us to formulate recommendations that take account of these characteristics.

B.1 The Inhalation of Asbestos Fibres

The importance of fibres in terms of their health effects following inhalation depends principally upon the following factors: (i) penetration — the ability of fibres to enter the respiratory system; (ii) deposition — where fibres are deposited within the respiratory system; (iii) retention — the extent to which fibres may be retained at the site of deposition; (iv) removal — the rate of clearance of fibres from the respiratory system; (v) translocation — the movement of fibres to other sites within the body; (vi) possibly durability — the ability of fibres to survive for some period of time rather than being dissolved or breaking up in tissue; and (vii) the biological effects of the fibres.[2]

Whether or not inhaled asbestos fibres will enter the lung depends upon the aerodynamic behaviour of the particles, the dimensions of the respiratory tract they enter, and the breathing patterns of the individual.[3] Dr. Victor Timbrell and his colleagues at the Medical Research Council Pneumoconiosis Unit at Penarth, Wales, have demonstrated that the aerodynamic behaviour of particles or their settling rate is a function mainly of the diameter of the fibre.[4] Dr. Timbrell has shown that the maximum diameter of respirable particles is between 3 and 3.5 microns.[5]

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[2] Graham W. Gibbs and Chung-Yung Hwang, "Physical Parameters of Airborne Asbestos Fibres in Various Work Environments — Preliminary Findings," *American Industrial Hygiene Association Journal* 36:6 (June 1975): 459–460; Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 96; Victor Timbrell, "The Inhalation of Fibrous Dusts," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 255–273; and Victor Timbrell, "The Inhalation of Fibres," in *Pneumoconiosis: Proceedings of the International Conference, Johannesburg, 1969*, ed. H.A. Shapiro (Cape Town: Oxford University Press, 1970), pp. 3-9.

Once fibres enter the respiratory system, three factors come into play: (i) Diameter — long fibres, if thin enough, can avoid being deposited by gravitational settlement or inertial precipitation in the nasal passages or high in the respiratory tract and may travel deeply into the lung.[6] (ii) Length — the length of the fibre also influences its chance of being intercepted high in the tracheal-bronchial tree before it can be deposited in the lung. (This is because the airway passages along which it must travel are fairly narrow. For this reason, very long fibres, that is, fibres over 100 microns in length, are unlikely to be distributed into the more peripheral or terminal air spaces of the lung.[7] Examination of pathological specimens by electron microscopy indicates that there are virtually no fibres longer than 200 microns in length in the lung and most are less than 50 microns in length.)[8] (iii) Shape — to the extent that chrysotile fibres are curly in shape, they correspondingly have a greater cross-sectional area and therefore interception at airway branches and in nasal passages is more likely. A curly chrysotile fibre would tend not to get down as deeply into the lung as a straighter, more needle-like amphibole fibre.[9]

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[3] Margaret R. Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," *American Review of Respiratory Disease* 114:1 (July 1976): 193.

[4] Timbrell, "The Inhalation of Fibrous Dusts," p. 273. Dr. Timbrell demonstrated that the important parameter for the main deposition mechanisms is particle free-falling speed, and for a fibre this is predominantly determined by the diameter and not the length of the fibre. This in turn explains the presence of asbestos fibres 50 microns and even 200 microns in length in the lung. See also, RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 97.

[5] Ibid. Fibres greater than 3 to 3.5 microns in diameter will not penetrate into the alveolar region of the lung.

[6] Timbrell, "The Inhalation of Fibres," p. 3. Dr. Timbrell explained ". . . that the free-falling speed of a fibre is approximately proportional to the diameter squared and almost independent of the length. Only if a fibre is of small diameter can it avoid deposition from gravitational settlement and inertial impaction high in the respiratory tract and succeed in penetrating to the pulmonary air spaces."

[7] Timbrell, "The Inhalation of Fibres," p. 3; and RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 96. As Dr. Gibbs explained, as fibres try to pass through airways which are fairly narrow, a fibre which is fairly long and gets out of orientation so that it is angled to the wall will be trapped. Also, if there is a change in direction of airflow, as there would be at a branch, the momentum of the fibre may carry the fibre onto the branch.

For those asbestos fibres that are deposited within the lung, the body has a fairly efficient defence system to deal with them. Two main biological mechanisms participate in the clearance of the fibres from the lower respiratory tract: (i) Most of the dust containing asbestos fibres is removed by the mucociliary escalator of the tracheal-bronchial tree. The material is either coughed up or swallowed. In general, it would appear that short fibres are cleared more readily by this method than long fibres.[10] (ii) Certain cells have the responsibility for defence of the host against all alien materials and these cells produce their effect by their ability to engulf foreign material. These phagocytic, that is, scavenger cells are known as macrophages. Depending upon what the foreign material is, the macrophage, by liberating enzymes within itself, can digest or destroy the material. In instances where the macrophage is incapable of destroying the material, as is the case with asbestos, the macrophage can keep the foreign substance from doing any harm by simply engulfing it.[11] It is also thought that the coating of asbestos fibres (asbestos bodies) may occur when the fibres have been engulfed by the macrophages.[12]

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[8] Paul Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," in *Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods*, Gaithersburg, Maryland: 18–20 July 1977, NBS Special Publication 506 (Washington, D.C.: U.S. National Bureau of Standards, November 1978), p. 136; Timbrell, "The Inhalation of Fibrous Dusts," pp. 269ff.; and Marc Trudeau, "Methods for the Evaluation of Asbestos Dust Concentration at the Workplace," in Mineralogical Association of Canada, Université Laval, Québec: 20–22 May 1979, *Short Course in Mineralogical Techniques of Asbestos Determination*, ed. R.L. Ledoux (Toronto: Mineralogical Association of Canada, Royal Ontario Museum, May 1979), p. 215.

[9] See Timbrell, "The Inhalation of Fibres," p. 6, where the author stated that "Curvature in a fibre therefore works to decrease the efficiency of penetration, especially in narrow airways. Looked at in another way, introduction of curvature into a fibre causes deposition to be earlier and the site to be higher in the respiratory tract. The fibres most affected are the long fibres, which are also in general the most massive." See also, Gibbs and Hwang, "Physical Parameters of Airborne Asbestos Fibres in Various Work Environments — Preliminary Findings," p. 459; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 45. Dr. Gibbs is of the view that the curliness of the chrysotile fibre has been somewhat overemphasized. His examination of the airborne fibres that exist in the mines and mills indicated that there was some curliness to the chrysotile fibre but not nearly as much as in the UICC (Union Internationale Contre le Cancer) samples used by Dr. Timbrell. RCA Transcript, Evidence of Dr. Graham W. Gibbs, 13 November 1981, Volume no. 33, pp. 20–21.

[10] RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 19–22; John E. Craighead and Brooke T. Mossman, "The Pathogenesis of Asbestos-Associated Diseases," *The New England Journal of Medicine* 306:24 (17 June 1982): 1448; and Telephone communication between Dr. Jerrold Abraham, Department of Pathology, College of Medicine, State University of New York, Syracuse, New York and Royal Commission on Asbestos Staff, 18 August 1983, together with confirming letter from Royal Commission on Asbestos Staff to Dr. Abraham of same date. Dr. Abraham and Dr. Cecilia Smith, University of California at San Diego, conducted an animal experiment in which rats were exposed to an aerosol of chrysotile taken from the Jeffrey Mine in Quebec. An analysis of the fibres recovered from the lungs of these rats at subsequent time intervals indicated that the short fibres tended to clear the lungs while the long fibres remained.

Shorter fibres, especially those less than 5 microns in length, are readily and completely engulfed by macrophages, but longer fibres are not; that is, those fibres longer than 5 microns or perhaps 8 microns in length are not completely engulfed by macrophages.[13] The importance of this in terms of disease production will be discussed below.

Macrophages are particularly mobile, and once these scavenger cells pick up asbestos fibres they follow one of three pathways. One pathway is to escape to the mucociliary apparatus, eventually to be coughed up and removed from the lung. A second pathway appears to be into the lymphatic system from which the cells are also removed. A third pathway is to penetrate deep into the tissues of the lung, and this appears to be the dangerous pathway from the point of view of disease production. The evidence is not clear as to why the macrophage chooses one pathway over the other.[14]

It has been estimated that the body's clearance mechanisms are 95 to 98% effective in removing asbestos fibres that have been inhaled into the lung.[15] There is no reliable evidence to indicate whether this clearance rate will vary in accordance with the manner of asbestos exposure. But the possibility does remain that high doses over short periods of time (peak exposures) or indeed continuous, higher levels of exposure will overwhelm the lung's clearance mechanism.[16] A variety of external influences such as cigarette smoke and air pollutants no doubt also affect the lung's clearance mechanisms, but these factors are difficult to assess, in part because individuals appear to differ in their response to inhaled particles. It is also difficult to assess the durability of fibres within the lung. It has been suggested by some authorities that either the ability of chrysotile to separate into tiny fibrils or its tendency to dissolve in weakly acidic environments might make it easier to be cleared from the lungs than the amphibole fibres.[17] Whatever the factor or factors of importance, there does appear to be evidence to indicate a preferential clearance of chrysotile as opposed to amphibole fibres from body tissue.[18]

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[11] RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 19–22; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 7–12.

[12] RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 16–17; and Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," p. 138.

[13] Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 194; RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), p. 24; and A. Morgan, "Effect of Length on the Clearance of Fibres from the Lung and on Body Formation," in *Biological Effects of Mineral Fibres*, vol. 1, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), p. 330.

[14] RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 20–22; and RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), p. 27.

[15] Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 194; and RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 38–39. It has been suggested that there are two phases of clearance through the tracheal-bronchial tree: about half the asbestos is removed within a few days; subsequently, clearance continues for extended periods. The bulk of this material is excreted in the feces. See Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," pp. 1448–1449.

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[16] RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, pp. 46–47. Dr. Becklake thought it likely both on the basis of scattered evidence and on the basis of what is known about lung clearance mechanisms that large, intense doses might permit greater temporary retention of asbestos fibres. See also, RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 41–42; RCA Transcript, Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41, p. 54; and RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), p. 36. Dr. Kotin was of the view that there is a difference biologically in the way one gets a dose of asbestos. He testified that it matters whether one gets a certain amount of dose in a very short period of time, as opposed to the same dose over a longer period, due to the possibility of overwhelming defence mechanisms in the body. See also, RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 61–62. Dr. Weill indicated it is possible that peak exposures will produce more disease because there may not be enough macrophage cells to clear the lung. Dr. John M.G. Davis et al. examined the effects of intermittent, high asbestos exposure (peak dose levels) on the lungs of rats. Their findings appear to refute the idea that very high exposures to asbestos for short periods result in a much higher lung burden of retained dust than might be expected. However, Dr. Davis in his testimony observed that in the experimental situation it was impossible to examine the effects of the realistic differential that one sees in factory situations. In his animal experiments he had only a fivefold difference between "even" and "peak" doses, whereas a peak dose caused by a temporary machine defect in a factory could easily be 100 times higher than the ordinary level. See John M.G. Davis et al., "The Effects of Intermittent High Asbestos Exposure (Peak Dose Levels) on the Lungs of Rats," *British Journal of Experimental Pathology* 61 (1980): 272–280; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 103–104.

[17] Dr. Alison D. McDonald suggested in her evidence that the amphiboles tend to remain as longer fibres once they get into tissue, whereas chrysotile does tend to split transversally into much shorter pieces. RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 47–48. Dr. Gibbs expressed the view that durability, that is, the ability of the fibre to survive for some period of time prior to either dissolving or breaking up into the tissue, is important. He noted that chrysotile, unlike the amphiboles, is not very acid resistant and that the ability to break apart is far greater for chrysotile than it is for the amphiboles. He suggested these differences might improve the ability of chrysotile to be cleared from the lung more efficiently than the amphiboles. RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 126–132. Dr. Eric J. Chatfield indicated that a fibre of an amphibole which goes into the lung is going to remain there as an amphibole fibre. However, the chemistry of a chrysotile fibre gives it the potential to be dissolved in the lung. RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, pp. 66–67. See also, RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 55–56.

B.2 The Ingestion of Asbestos Fibres

While inhalation is the principal method by which asbestos fibres enter the body, they may also enter through ingestion, either in food, beverages, drinking water, or drugs. In contrast to inhalation, there is no limitation on either the length or diameter of an asbestos fibre that is capable of being ingested. In theory, fibres of any size or shape might be present in food or beverages or drinking water and therefore capable of being swallowed; in practice, the sizes of fibres that are ingested are typically much smaller than those that are inhaled, averaging around 2 microns in length.[19]

It would appear that fibres entering the body through inhalation and ingestion are dealt with quite differently. Fibres which are swallowed tend to pass right through the gastrointestinal tract and be immediately removed. Fibre penetration of the tissue or lining of the gut following ingestion is speculative at best.[20] On the other hand, fibres which are inhaled and are too long to be engulfed by macrophages are not removed but remain in the body and accordingly have ample opportunity to penetrate the tissue of various organs.

B.3 The Pathogenesis of Asbestosis

Fibrosis is the formation of fibrous or scar tissue, usually as a reparative or reactive process. It was originally thought that the fibrogenic effect of asbestos fibres was due either to the cells in the lungs being physically irritated by the fibres or to the insolubility of fibres in tissue.[21]

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[18] Margaret R. Becklake, "Exposure to Asbestos and Human Disease," *The New England Journal of Medicine* 306:24 (17 June 1982): 1481.

[19] See Chapter 11 of this Report. See also, RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, p. 97.

[20] See Chapter 5, Section 1 of this Report. See also, R.E. Bolton and J.M.G. Davis, "The Short-Term Effects of Chronic Asbestos Ingestion in Rats," *Annals of Occupational Hygiene* 19:2 (November 1976): 121-128; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 97-100. Dr. Davis testified that he has examined the gastrointestinal tissue and other tissue of animals that had ingested asbestos for their full lifespan and could not find any asbestos, let alone any gastrointestinal or other tumours. See also, J.M.G. Davis, R.E. Bolton, and J. Garrett, "Penetration of Cells by Asbestos Fibers," *Environmental Health Perspectives* 9 (1974): 255-260; and World Health Organization, International Agency for Research on Cancer Working Group, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, vol. 14: *Asbestos*, Lyon, France: 14-17 December 1976 (Lyon: IARC, 1977), pp. 59-60.

[21] Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," p. 139.

Recent studies have led to other hypotheses based on the response of the macrophage. Asbestos fibres totally engulfed within the macrophage are relatively immobile and not likely to be harmful. If, however, the fibre is liberated, then either the fibre or the process by which it is liberated may produce an adverse health effect. Fibres attacked by macrophages may be liberated in one of two ways. First, a totally engulfed fibre will survive within the macrophage and the macrophage will try to destroy the fibre by liberating within itself certain enzymes. As contrasted with inorganic materials, these enzymes are not successful in destroying the fibre. Macrophages have a life of 30 to 40 days, and when the macrophage dies the engulfed fibre is freed into the surrounding medium.[22] It may subsequently be re-engulfed by other macrophages. A second way in which the fibre becomes liberated arises if it has a length greater than that of the macrophage itself. Then, the macrophage will be physically incapable of ingesting the entire fibre and the fibre will only be partly engulfed by the macrophage. The macrophage defence system does try to respond to these longer fibres. In circumstances where macrophages cannot completely engulf fibres, the secretion or release of enzymes or other substances may be greater than when the macrophage can completely engulf the fibre. With a long fibre, many macrophages may accumulate and may fuse to form a giant cell.[23]

Beyond this point the pathogenesis of asbestosis is not at all well understood, but one theory has it that the enzymes which leak from incompletely engulfed cells dissolve the protein in the lining of the alveolar sacs, leading to tissue death and fibrosis.[24] Another hypothesis is that the enzymes and other substances released by the macrophages stimulate the proliferation of another type of cell called a fibroblast. In turn, fibroblasts contribute to fibrosis by synthesizing increased amounts of connective tissue such as collagen.[25] One further hypothesis is that some of these fibroblasts actually engulf asbestos fibres in their own right. It is quite possible that asbestos stimulates the production of collagen by various cells within the body, such as fibroblasts, thereby causing fibrosis.[26]

Another way in which the body reacts to asbestos fibres, the importance of which is not entirely clear, is by the formation of asbestos bodies.

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[22] RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 25-31.

[23] Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," p. 1449; RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982. Volume no. 34, pp. 7-12; and RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 25-31.

[24] RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 28-31.

[25] Ibid. See also, Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," p. 1449; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 12-15.

[26] RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 12-15, 25.

These bodies are believed to be formed by macrophages that have engulfed fibres.[27] Only a small percentage of all fibres inhaled into the lung do become coated, and it is not known why most of the fibres are not coated. The presence of asbestos bodies does not appear to stimulate fibrosis, and in fact there is evidence that tends to suggest that the coating of fibres renders them at least non-fibrogenic.[28] However, approximately three-quarters of all asbestos fibres remain uncoated. These fibres tend to accumulate in the peripheral regions of the lower lobes of the lungs, the site of early fibrosis.[29] Over time, the fibrotic process progresses in an apparently diffusive manner, affecting more and more of the lung. As the fibrosis becomes extensive, the lung shrinks to below normal size and in cases of advanced asbestosis may take on a "honeycomb" appearance.[30] Figure 4.1 shows a typical lung with fibrosis.

B.4 Carcinogenesis

What are the mechanisms of asbestos-induced carcinogenesis in humans? The short answer to this question is that the scientific and medical community appears to know very little about these mechanisms. It is thought that asbestos-induced cancer, like other types of cancer, originates from cells which have been transformed, frequently by changes in or damage to the DNA or other genetic material.[31] Although this damage to the DNA may be repaired, permanent cell transformation may occur if the repair mechanisms are ineffective or if the damage is not repaired correctly.[32] In the case of asbestos, the target cells that appear to be trans-

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[27] Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," p. 138.

[28] Ibid. See also, RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 16–17.

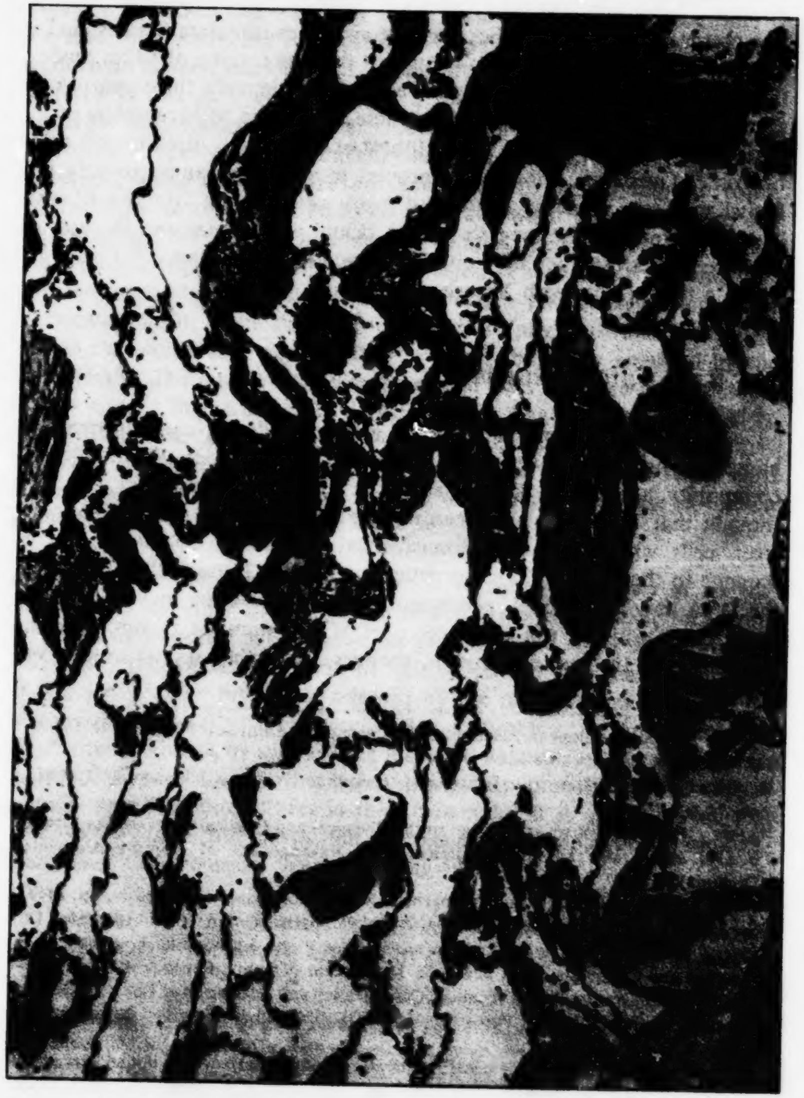
[29] Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," p. 138; and Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," p. 1449.

[30] Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," p. 1449.

[31] See generally, Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," pp. 1446–1455; John Cairns, *Cancer: Science and Society* (San Francisco: W.H. Freeman and Company, 1978); and U.S., Department of Labor, Occupational Safety and Health Administration, "Identification, Classification and Regulation of Potential Occupational Carcinogens," 29 CFR Part 1990, 45 FR 5002–5296, 22 January 1980 [hereafter "The OSHA Cancer Policy"].

[32] There is some suggestion in the scientific literature that carcinogenesis is not an irreversible process necessarily, so that even though the initial interaction with the DNA has taken place, the cells are capable of being repaired by a series of repair mechanisms available to the genetic material. RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 113–114; RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, p. 92; and Cairns, *Cancer: Science and Society*, pp. 53, 85ff. However, it cannot be concluded that the repair mechanisms are completely reliable. Indeed, many authorities believe that DNA repair is not normally 100% efficient. See "The OSHA Cancer Policy," pp. 5124–5127.

Figure 4.1
Photomicrograph of a Lung Showing the Changes Typical of Asbestosis*



Note: *Picture shows patchy fibrosis thickening some of the alveolar walls, while others remain normal, fine, and delicate.

SOURCE: Picture and letter from Dr. Alexander C. Ritchie, Professor of Pathology, University of Toronto to the Royal Commission on Asbestos, 4 October 1983.

formed are the epithelial cells of the bronchial tree and the gastrointestinal tract and the mesothelial cells of the pleura and peritoneum. There appears to be considerable evidence that at least some cancers may originate from a single transformed cell, but it is not entirely clear whether the asbestos-induced cancers fall into this category.[33] The early cellular and molecular events of the disease and the mechanisms which initiate it are not well understood.[34]

The asbestos-related cancers, like other cancers, are characterized by progressive cellular changes through a number of stages, leading to the development of abnormal or transformed populations of cells. Many cancer experts believe that a variety of different factors can act to initiate or accelerate the development of cancer at each stage and that often these factors will interact with each other, producing a synergistic effect.[35] That asbestos can act as a carcinogenic agent in its own right is strongly suggested by its effect in producing mesothelioma because the mesothelial tissues are not directly exposed to other environmental carcinogens.[36] That asbestos can also serve as a carcinogen by promoting or accelerating multiplication of malignant cells is strongly suggested by its effect in producing lung cancer in conjunction with cigarette smoking.[37] But whether asbestos fibres act in this way because of their physical dimensions, their inherent or acquired chemical composition and structure, or because of the effects of factors released or formed by their interaction with macrophages is again uncertain.

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[33] "The OSHA Cancer Policy," pp. 5023–5024; Kenny S. Crump et al., "Fundamental Carcinogenic Processes and Their Implications for Low Dose Risk Assessment," *Cancer Research* 36 (September 1976): 2973; and Stephen Shugar, *Effects of Asbestos in the Canadian Environment*, prepared for the NRC Associate Committee on Scientific Criteria for Environmental Quality, NRCC, no. 16452 (Ottawa: National Research Council Canada, 1979), p. 134. If cancer can develop from a single cell, that cancer can be initiated by the interaction of a single molecule of a carcinogen with the critical target site in a cell. This does not necessarily mean that a person will develop cancer from the application of a single molecule of the carcinogen, since the probability of the interaction taking place appears to depend on the dose. Still, if a tumour can be traced back to a single molecular interaction, this suggests that any dose above zero may have an effect, and there is unlikely to be a threshold dose below which the induction of cancer cannot occur. See Chapter 5, Section E of this Report for a discussion of dose-response relationships.

[34] Craighead and Mossman have stated that "At present, the mechanism of asbestos-associated carcinogenesis is unclear, although the mineral appears to act like a classic tumor promoter. The fibrous nature of asbestos is critical . . . ." See Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," p. 1452.

[35] "The OSHA Cancer Policy," pp. 5020–5022.

[36] Irving J. Selikoff and Douglas H.K. Lee, *Asbestos and Disease* (New York: Academic Press, 1978), pp. 417–423.

[37] Ibid. See also, Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," p. 1451.

Not only is there uncertainty as to how asbestos causes tumours at the target site, it is even uncertain as to how asbestos fibres get to the site of the tumour in the first place. In the case of pleural mesothelioma, it is thought likely that some of the asbestos deposited in the lung tissue reaches the pleura either by lymphatic transport within macrophages, or by the direct penetration of free fibres, or through the bloodstream.[38] Once positioned in the pleura, some of the fibres evidently are able to cause the development of mesothelioma, but the reasons for that development are not known. Similar uncertainty surrounds the development of peritoneal mesothelioma. It has been suggested by some authorities that asbestos fibres in the lungs are transported to the abdomen through the lymphatic channels of the body.[39] Once asbestos has entered the abdomen, it is thought that the mechanisms of carcinogenesis of the pleural and peritoneal cavities are similar.[40] But, again, the mechanisms of the malignant transformation of the mesothelial tissues remain obscure.

The same may be said for the development of asbestos-induced lung cancer. It was originally thought that this tumour was a scar cancer stimulated by the presence of asbestosis in the lungs because of its common occurrence in association with fibrosis and because of its common location in the lower lobes of the lung where fibrosis tends to be most marked.[41] However, reported cases of asbestos-induced lung cancer, either in association with very mild fibrosis or in its absence altogether, have left the reasons for the development even of this tumour unresolved.[42]

The way in which asbestos reaches the gastrointestinal tract so as to induce alimentary tract tumours is also far from firmly established. One possibility is that the coughing up and swallowing of fibres following inhalation is responsible.[43] Another possibility, suggested by Dr. John M.G. Davis in his testimony before this Commission, is that asbestos fibres after inhalation have the ability to move around the body by the lymphatic channels to various sites, including the gastrointestinal tract.[44] Dr. Alexander C. Ritchie, Professor of Pathology at the University of Toronto, expressed serious reservations about this possibility in his testimony before this Commission. Dr. Ritchie had no doubt that fibres find their way to the gastrointestinal tract, but he candidly observed that we just do not know how they get there.[45]

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[38] RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), p. 78; John M.G. Davis, "The Biological Effects of Mineral Fibres," *Annals of Occupational Hygiene* 24:2 (1981): 228; and Personal communication between Professor Patrick Sebastien, Institute of Occupational Health and Safety, McGill University, Montreal, Quebec and Royal Commission on Asbestos Staff, 21 April 1983.

[39] Craighead and Mossman, "The Pathogenesis of Asbestos-Associated Diseases," p. 1450.

[40] Ibid.

[41] RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, p. 77; and Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 214.

[42] Dr. J. Corbett McDonald testified before this Commission that his data are compatible with most excess lung cancers having occurred in people who had x-ray abnormalities. But he also indicated that there is evidence that some of the excess lung cancers he has observed are not explained in that way. In his view, there is no special reason to believe that the two processes of asbestosis and lung cancer are produced by the same mechanisms. RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 37. Dr. Becklake suggested there may be two different cell types of cancer attributable to asbestos: one which relates to squamous cell cancer in the major airways, and one which is a true scar cancer and therefore likely to be related to or stimulated by the presence of fibrosis in the lungs. RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, p. 77.

The carcinogenic effects of asbestos do appear to have certain distinctive features in common with the effects of other carcinogens. For one thing, the effects of asbestos appear to be irreversible, not in the sense that cells transformed by contact with asbestos will inevitably develop into cancer, but that they will retain their potential to do so.[46] This means that humans exposed to asbestos will generally remain at increased risk of developing cancer even after exposure to asbestos has ceased. A second distinctive feature of asbestos-related cancers is that they are age-dependent.[47] Cancers in general tend to occur among older age groups, and asbestos-related cancers are no different.[48] A final feature that asbestos-related cancers share in common with other cancers is one we have already observed in this Report: there is normally a latency period of many years between time of first exposure and the clinical manifestation of the disease.[49]

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[43] Eric J. Chatfield, "Measurement of Asbestos Fibres in the Workplace and in the General Environment," in *Short Course in Mineralogical Techniques of Asbestos Determination*, p. 114; Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Asbestos Fibers," p. 138; and Anthony B. Miller, "Asbestos Fibre Dust and Gastrointestinal Malignancies," Appendix 5 in The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981.

[44] RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 100-101. Dr. Davis contrasted the results of animal inhalation experiments and animal ingestion experiments. Following the former he was able to find asbestos fibres scattered all around the animals' bodies, including the gastrointestinal tract; following the latter he could not find any asbestos in the gastrointestinal tissue of animals.

[45] RCA Transcript, Evidence of Dr. Alexander C. Ritchie, 14 July 1982, Volume no. 49, pp. 14-19, 31-35. Dr. Ritchie testified that in his opinion it was most improbable that asbestos fibres reached the gastrointestinal tissue by the lymphatics. He indicated that the lymph flow from the stomach to the lung is from the stomach upwards, so that one has to imagine the asbestos fibres swimming against the flow. Even if one allows that the fibres could proceed against the normal direction of flow, on the ordinary view that the dangerous fibres are the long, thin ones, then the fibres would be in little vessels that are really not much bigger than the fibres themselves and certainly much narrower than 10 microns in length. Dr. Ritchie himself preferred "the bloodstream to the lymph stream" as the more likely mechanism by which fibres are transported to the gastrointestinal system. But he went on to indicate that both the bloodstream and direct passage through the tissues seem to him to be improbable mechanisms. He did, however, testify that from two cases he had personally examined, he knew that asbestos fibres can be found in the tissues in and around gastrointestinal carcinomata.

[46] "The OSHA Cancer Policy," p. 5024.

[47] Ibid., p. 5026.

[48] For a more detailed discussion of the age- and time-dependence of asbestos-related cancers, see Chapter 5, Section E of this Report.

One final matter deserves mention. Whether any particular individual will develop a malignant tumour, the amount of exposure necessary for the initiation of carcinogenesis, and the rate of growth and spread of a tumour, once initiated, appear to depend upon a number of factors all of which may be subsumed under the term "individual susceptibility."[50] The efficiency of an individual's lung clearance mechanism, the anatomic characteristics of his lung and airway system, the physical fitness of the individual, and the nature of his immune system all suggest that some individuals are more susceptible to the development of cancer than others.[51] There is no concrete evidence which identifies individual susceptibility in relation to asbestos exposure and either cancer or fibrosis, but common sense and experience tell us that such individual susceptibility does indeed exist.[52]

C. Principal Sources of Information on the Health Effects of Asbestos

The evidence on the etiology of asbestos-related diseases is derived chiefly from three sources: (i) the human experience, as measured by case reports and epidemiological studies of defined populations; and more limited post-mortem studies which analyze by electron microscopy the asbestos fibre content of lung tissue; (ii) experimental work with animals; and (iii) *in vitro* or cell culture studies.

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[49] "The OSHA Cancer Policy," p. 5026. But the traditional theory of latency, at least in respect of asbestos-related diseases, has recently been challenged. See Chapter 5, Section E of this Report.

[50] RCA Transcript, Evidence of Dr. Alexander C. Ritchie, 14 July 1982, Volume no. 49, pp. 40-41. However, Dr. Ritchie knew of no evidence which identifies susceptibility in relation to asbestos exposure and cancer. See also, Philip E. Enterline, "Pitfalls in Epidemiological Research: An Examination of the Asbestos Literature," *Journal of Occupational Medicine* 18:3 (March 1976): 150-151; and "The OSHA Cancer Policy," pp. 5129-5138.

[51] Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," pp. 190-191.

[52] Dr. William J. Nicholson testified that the Mount Sinai School of Medicine in New York is beginning to look at the possibilities of identifying individuals at particularly high risk among those equally exposed; in other words, what means exist to predict who would be likely to get cancer and who would not. The possibility exists that this can be done by measurement of immune competence. RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 134. See also, Elliott Kagan, "Immunological Status and Host Resistance to Asbestos-Related Disease," in *Proceedings of the World Symposium on Asbestos*, Montreal, Quebec: 25-27 May 1982 (Montreal, P.Q.: Canadian Asbestos Information Centre [1983]), pp. 442-448. The author indicated that research is being done in the area of sophisticated immunological probes to detect persons "at risk" of developing asbestos-related cancers.

Given the uncertainty that exists about many of the most important health issues concerning asbestos, the available evidence from the various studies and tests requires careful assessment. That assessment can best be done within the framework of an accepted set of principles that will enable us to interpret and weigh properly the health evidence before us. For such a set of principles, we again turn to Dr. Sackett and to the tests for causation articulated by him.[53] Those tests particularly relevant to studies of the association between asbestos exposure and disease (listed in descending order of importance) are as follows:

(i) Is there a strong association between exposure and health outcomes of interest? "Strong" here means the odds favouring the outcome of interest with, as opposed to without, exposure to asbestos. The weaker the association, the greater the need for caution in relying upon the result. A fivefold increase in the incidence of lung cancer among an asbestos-exposed population would be strong evidence of a relationship between asbestos exposure and the disease. A twofold increase would have to be treated more cautiously.

(ii) Is the association consistent from study to study? The repetitive demonstration by different investigators studying different populations of an association between exposure and the outcome of interest gives consistency and thus credibility to the association.[54] For example, much of the credibility concerning the causal link between smoking and lung cancer arises from the repeated demonstrations of a strong statistical association in case-control, cohort, and other studies.

(iii) Is there a dose-response relationship? As it is an accepted biological principle that the greater the dose the greater the response, studies which demonstrate not simply a response but also a response that increases with dose provide greater support for a causal relationship. This is a crucial issue and is considered at length in Chapter 5 of this Report.

(iv) Does the association make epidemiological sense? Are the investigator's results in agreement with current understanding of the distribution of causes and health outcomes in humans?

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[53] Sackett, "A Review of Diagnostic Tests for Causation," pp. 10-16 and Appendix B, *passim*. These tests were originally articulated by Austin Bradford Hill, *Principles of Medical Statistics*, 9th ed. (London, England: Oxford University Press, 1971), p. 320. See also, J. Corbett McDonald, "Asbestos and Lung Cancer: Has the Case Been Proven?" *Chest* 78:2 (August 1980, Supplement): 374-376; and Austin Bradford Hill, *A Short Textbook of Medical Statistics* (London, England: Hodder and Stoughton, 1977), pp. 285-296.

[54] RCA Transcript, Evidence of Mr. Richard A. Lemen, 8 July 1981, Volume no. 17, p. 84.

(v) Does the association make biological sense? Is it consistent with current understanding of the responses of human cells, tissues, and organs to the postulated causative agent or agents?

(vi) Is the association specific? Is the association limited to a single putative cause and a single effect, or is the association clouded by the possibility of multiple causes and overlapping effects? Asbestosis and mesothelioma have a quite specific association with asbestos exposure. On the other hand, lung, laryngeal, and gastrointestinal cancer, which can be associated with asbestos exposure, are also associated with smoking, diet, and lifestyle, thus clouding the association.

(vii) Is the association analogous to a previously proven causal relationship? This test links, for example, asbestosis among asbestos workers who inhale dust particles containing asbestos to the earlier known causal relationship between silicosis and the inhalation of dust particles containing silica.

Not all of these tests are applicable to every health issue before us, nor need each test be satisfied in order to demonstrate a particular causal relationship. Rather, proof of an association will depend upon the weight of the available evidence.

D. The Human Experience: Epidemiological Studies

Epidemiology is concerned with the study of the distribution and determinants of disease prevalence in humans. Asbestos has been the subject of a number of epidemiological studies which are reviewed in detail in Chapter 5 of this Report. The overwhelming importance of epidemiological data is that they provide evidence in humans of associations between exposures to various substances and patterns of disease. Specifically — and to the extent the necessary data are available — they permit the direct measurement of the risk of cancer or fibrosis due to exposure to asbestos in human populations.

The true experiment with humans, the so-called randomized trial, is the optimal epidemiological method to explore questions of causation. In the case of asbestos, the randomized trial is neither feasible nor ethical. This rules out a true experiment that would deliberately expose a randomly selected group of individuals to given doses of asbestos and compare their health outcomes to a randomly selected control group. Three other epidemiological methods are, however, available to examine the human disease experience with asbestos: the cohort study, the case-control study, and the case-series study. Of these, the most powerful analytical technique is the cohort study.[55]

D.1 The Cohort Study

The essential design of a cohort study is to define a group of persons exposed to a particular hazard (the cohort) and a group not so exposed (the control group). The experience of these two groups is followed over time and, where appropriate records are available, the morbidity and mortality experiences of the two groups are compared.

Typically, cohort studies may be retrospective or prospective. The cohort studies of asbestos-exposed populations published to date have been retrospective, so that what is being assessed is the past health experience of a group of workers exposed to asbestos. The fact that asbestos cohort studies are retrospective may impose limitations on their quality in that the epidemiologist can only use what is available in terms of exposure data, work histories, and the like. An asbestos mortality study might attempt to compare the observed number of deaths attributable to lung cancer in the selected cohort with the expected number of deaths due to lung cancer in the control group. The calculation of the expected number of deaths is made by reference to a more general population base, assumed not to be exposed to asbestos, be it the country as a whole, or the province or state where the cohort is located. This calculation of the expected number of deaths is standardized to take account of the death rates in specific age groups and in specific calendar time periods. The epidemiologist may also control for sex, race, and other factors to make the comparison more precise. In the result, the epidemiologist obtains a ratio of the observed number of deaths in the cohort to the expected number of deaths. This ratio is known as a standard mortality ratio (SMR), which is a measure of relative risk. An observed/expected ratio for lung cancer of 2, or an SMR of 200, means that the actual deaths from lung cancer in the cohort exposed to asbestos is twice the number that may be expected in the ordinary population not exposed to asbestos. An alternate way of saying the same thing is that the excess risk of lung cancer in the cohort is 100%. Studies which attribute death to mesothelioma or asbestosis do not generally provide an expected number of deaths calculation for the simple reason that deaths from these two causes are so rare in the general population (or control group) as to be zero for practical purposes.

Most asbestos cohort studies provide some quantitative data on exposures, in order that observed/expected ratios may be calculated at different exposure levels. Such information is necessary to determine dose-response relationships and to assess the incidence of disease in the cohort in terms of the severity of exposure. The common method of quantifying asbestos exposure in order to assess health risks has been to calculate exposures both in terms of the concentration at a given time and duration of exposure over time. The result, a measure of cumulative exposure, has been expressed in either million particles per cubic foot-years (mppcf-yrs) or fibres per cubic centimetre-years (f/cc-yrs), depending upon whether what has been measured were particles of dust or asbestos fibres. One fibre per cubic centimetre-years represents exposure to an average workday concentration of 1 fibre per cubic centimetre (f/cc) for 1 year; 2 fibres per cubic centimetre-years would represent either 2 years of exposure at 1 f/cc or 1 year of exposure at 2 f/cc. Similarly, 8 fibres per cubic centimetre-years might represent 1 year of exposure at 8 f/cc, 2 years of exposure at 4 f/cc, or 4 years of exposure at 2 f/cc. Where a cohort study does not have available quantitative measurements, duration of exposure is generally used as a surrogate for cumulative dose.

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[55] Sackett, "A Review of Diagnostic Tests for Causation," pp. 11-12 and Appendix B, pp. 2-4.

There is in cohort analysis the possibility of sampling variation or random error in measurement, which raises concerns that the standard mortality ratio (or relative risk) may indicate that there is an adverse health effect when in truth there is not; or may indicate that there is no effect when in truth there is. Accordingly, authors of most of the epidemiological studies have provided statistical confidence intervals to their calculations and have indicated where a calculation is statistically significant as opposed to being the possible result of chance.

D.2 Criteria for Evaluating Cohort Studies and the Limitations of Asbestos Epidemiology

Just as there exists a set of criteria for the general evaluation of health effects evidence, there is a separate set of recognized criteria for assessing the quality of cohort studies. Several of our expert witnesses made reference to these criteria during the course of their testimony.[56] We deem it useful to make reference to them in our Report for they both provide a framework for the critical appraisal of the epidemiological literature and enable us to focus upon the deficiencies in asbestos epidemiology. However unavoidable these deficiencies are, given the data that are available, their expression serves to emphasize the uncertainty that pervades the evidence on the health effects of asbestos.

For convenience, we group the relevant criteria under four headings: the study population; the quantification of exposure; mortality or morbidity ascertainment; and statistical analysis.

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[56] For a general discussion of these criteria, see the Appendix to Chapter 7 of this Report.

(a) The Study Population

Selection Criteria — To ensure that any effects of exposure will manifest themselves, a sufficient number of the study population should have a substantial amount of exposure. The study by Dr. J. Corbett McDonald et al. of the Quebec chrysotile miners and millers is likely the best in this regard for the authors identified everyone who had ever worked for one month or more in the mines and mills and who was born between 1891 and 1920.[57] We note that it may be disadvantageous to include only long-term employees because they may be a survivor population and hence may not give a true picture of the disease incidence in the cohort. The study by Dr. Philip E. Enterline and Ms. Vivian L. Henderson of Manville employees has been criticized because the authors selected only retirees, those over age 65, for inclusion in their cohort. On the other hand, as Dr. Enterline pointed out, there is an advantage to studying retirees. Since they are no longer being exposed, one can easily separate the effects of dose from the effects of duration of exposure.[58] Ideally, the epidemiologist would wish an even distribution of employees in terms of age, duration of exposure, and intensity of exposure. In practical terms, he can do no better than the actual distribution of the cohort he is studying.

Number of Subjects — The capacity of a morbidity or mortality study to detect excess risk is directly related to the size of the cohort and to the observed incidence of disease or number of deaths. A small cohort and a small excess risk may not demonstrate a statistically significant association.[59] The epidemiologist thus has to ensure an adequate number of individuals for study within the cohort to be able to detect with reasonable statistical confidence a true increase in the level of disease. The Quebec study of miners and millers (with over 11,000 members of the cohort and over 4,500 observed deaths), the Ferodo friction materials plant study (with over 13,000 members of the cohort and over 2,100 deaths), and the North

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[57] J. Corbett McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," *British Journal of Industrial Medicine* 37 (1980): 11-24.

[58] Philip E. Enterline and Vivian L. Henderson, "Type of Asbestos and Respiratory Cancer in the Asbestos Industry," *Archives of Environmental Health* 27:5 (November 1973): 316-317; and RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 38-40. For examples of criticisms of the use of a survivor population, see RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 30; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 83-85.

[59] See, for example, RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, pp. 32-34.

American insulators study (with 17,800 members of the cohort and over 2,200 deaths) offer potentially the strongest evidence.[60]

Of course, the maximum cohort size is invariably fixed by the number of past employees so that the investigator is bound by the available population and there are few large cohorts of asbestos workers. This gives rise to a serious limitation of asbestos epidemiology: it is likely not sensitive enough to detect excess risk directly or without extrapolation at the lower levels of exposure that currently prevail — that is, 2 f/cc and below — unless the effect is a very large one. According to Dr. Enterline, even if the excess risk at 2 f/cc is as high as 25%, meaning that the SMR is 125, that excess is not statistically detectable by epidemiological methods for the simple reason that a large enough study population is not available.[61]

Type of Asbestos Exposure — There may be no asbestos-related health issue more vexing or more important than whether different fibre types or dimensions differ in their health effects. The investigation of this issue can be facilitated in a situation where the entire study population has been exposed to a single fibre type and in a type of process likely to yield fibres of a given size. The study by Dr. J. Corbett McDonald et al. of the miners and millers in Quebec (chrysotile only); the study by Mr. Geoffrey Berry and Dr. Muriel L. Newhouse of the Ferodo friction materials factory workers at Derbyshire, England (chrysotile only); the study by Dr. John M. Dement et al. of the asbestos textile factory workers at Charleston, South Carolina (chrysotile only); and the studies of the war-time gas mask workers by Dr. Alison D. McDonald and Dr. J. Corbett McDonald in Canada (crocidolite only) and by Dr. J.S.P. Jones et al. in England (separate crocidolite and chrysotile exposures) are examples of studies that best meet this criterion.[62] The investigation of this issue can also be facilitated in a situation where a cohort can be divided into groups of employees whose exposure differed only in fibre type, fibre size, or type of process, thereby providing an internal comparison within the same working environment. The Enterline study is of some assistance here, in that part of his cohort was exposed to single fibre types and part to a combination of chrysotile and the amphiboles.[63] If, however, the cohort being studied has had a mixed fibre exposure as, for example, in the study by Dr. Murray M. Finkelstein of the asbestos-cement workers at Scarborough, Ontario,[64] it makes inquiry into the possible differential pathogenicity of different fibre types and fibre sizes that much more difficult. Unfortunately, few study populations or groups within study populations have been exposed solely to one fibre type, size, and process. Given that these factors may affect the disease rate, in a situation where these factors differ as between studies, it is difficult to sort out the most salient features.

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[60] J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75"; Geoffrey Berry and Muriel L. Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," *British Journal of Industrial Medicine* 40:1 (February 1983): 1-7; and Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 91-116.

[61] Ontario, Royal Commission on Asbestos, Exhibit II-3 [hereafter RCA Exhibit], in RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8: Philip E. Enterline, "Epidemiologic Basis for the Asbestos Standard," paper presented at the Second Annual Symposium on Environmental Epidemiology, Pittsburgh, Pennsylvania, 28 April 1981, Table 2. (Mimeographed.) See also, RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 26-33. Dr. Enterline addressed the question as to whether epidemiology can provide any evidence on the safety of the 2 f/cc standard. In his testimony, he offered the view that epidemiology might detect an excess risk of 50% or greater. Thus, for example, Dr. J.C. McDonald's study of the Quebec miners indicated a 6% excess risk after 50 years' exposure at 2 f/cc. According to Dr. Enterline, a study population of 220,000 would be required for that excess to be statistically detectable.

(b) The Quantification of Exposure

In light of our discussion thus far, the need for quantitative data on exposure levels is obvious. Here, three main factors come into play. First we list them and then discuss their limitations. A fourth factor, smoking data, is considered separately.

Work History — It is important that the epidemiologist have an accurate description of each individual's job history and where in the plant being studied each job was performed. Only then can he hope to utilize the measurements available for the plant in order to determine each cohort member's overall exposure.

The Quantity of Industrial Hygiene Data — This criterion relates to the number of measurements taken in the plant, where in the plant they were taken, and the years in which they were taken. In the absence of actual measurements, duration of exposure, which does not differentiate as to the level of exposure, must serve as an index of dose.

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[62] J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75"; Berry and Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos"; John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," *Annals of Occupational Hygiene* 26:1-4 (1982): 869-887; Alison D. McDonald and J. Corbett McDonald, "Mesothelioma After Crocidolite Exposure During Gas Mask Manufacture," *Environmental Research* 17 (December 1978): 340-346; and J.S.P. Jones et al., "The Consequences of Exposure to Asbestos Dust in a Wartime Gas-Mask Factory," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 637-653.

[63] Philip E. Enterline, Pierre DeCoufle, and Vivian L. Henderson, "Mortality in Relation to Occupational Exposure in the Asbestos Industry," *Journal of Occupational Medicine* 14:12 (December 1972): 900; and Vivian L. Henderson and Philip E. Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 122.

[64] Murray M. Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," *British Journal of Industrial Medicine* 40 (1983): 138-144.

The Quality of Industrial Hygiene Data — The quality of the data in asbestos epidemiology is basically related to questions of whether measurements were made in fibres or in dust particles, whether there were any side-by-side measurements, and whether there is any reliable relationship between particle counts and fibre counts in the cohort under review.

As we have indicated, cohort studies of asbestos workers by their nature are historical or retrospective rather than prospective: the studies were all undertaken long after the exposures occurred. The epidemiologist must work backwards in time to gather the necessary data for his study. It is therefore no accident that one of the weakest parts of all asbestos epidemiology is the quality of the quantitative exposure data. Although asbestos is perhaps the best studied carcinogen, next to radiation, the industrial hygiene data are seriously deficient.[65] In particular, we identify the following deficiencies:

(i) Work histories tend to be inaccurate because industry kept very little by way of detailed records. Even where work histories are available, the description of the position a worker held does not necessarily divulge what he did. Such exposure data as do exist are not refined so that there is little or no evidence on where an employee was in a plant and what hours of the day he was there, data which could be very important in determining his real exposure.[66]

(ii) There is a serious lack of measurements of any kind, particularly in the period before 1950. Those are precisely the years when exposures were higher and when by now a sufficient period has elapsed so that the disease effects of that exposure have become manifest. For example, Dr. Dement's study had over 5,000 individual measurements in the plant during the period 1960–1975, but less than 200 measurements in each of the periods 1930–1945 and 1945–1960.[67] While Dr. J.C. McDonald, in his Quebec study, had over 4,000 midget impinger readings available in the period 1949–1966, very few of these were in underground mines or open pits where the bulk of the cohort was exposed and there were virtually no readings of any kind in the pre-1949 period.[68] In the Scarborough plant studied by Dr. Finkelstein, measurement data exist for only four of the years between 1948 and 1960.[69]

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[65] RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 90.

[66] See, for example, Julian Peto, "Dose-Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards, Part II. Mortality," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 197. See also, the Appendix to Chapter 7 of this Report.

[67] Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," Table 1, p. 871.

(iii) Almost all of the measurements in the cohort studies now available were in terms of dust particles, whereas exposure to asbestos is now regulated in terms of fibres. This would not necessarily be a weakness if there was a reliable and accurate method of converting particle measurements to fibre measurements. Considerable efforts have been made by several epidemiologists to convert the dust measurements in their studies to fibre counts. On the basis of the testimony we heard concerning the results of these efforts, we can say generally that converting dust particles to fibres is an uncertain process.[70] Moreover, the conversion factors that apply, for example, in textiles are likely not applicable to those in mining or even in friction materials. Even the conversion factors for textile carding and spinning do not likely apply to other textile operations. Within a specific process, conversions may be fairly reliable.[71]

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[68] J. Corbett McDonald and F. Douglas K. Liddell, "Mortality in Canadian Miners and Millers Exposed to Chrysotile," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 1-9; RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 5; and RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 31. See also, the Appendix to Chapter 7 of this Report.

[69] Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory"; and RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 46-48.

[70] The process is sufficiently uncertain that, in their earlier publications, Dr. J.C. McDonald and his colleagues indicated that they could not express their results confidently in fibre counts. See J. Corbett McDonald et al., "The Health of Chrysotile Asbestos Mine and Mill Workers of Quebec," *Archives of Environmental Health* 28:2 (February 1974): 68. In their later work, Dr. J.C. McDonald et al. attempted to convert dust particle measurements from the Quebec mines to fibre measurements. In his oral testimony before this Commission, Dr. J.C. McDonald commented on their efforts as follows:

> It's more or less fizzling out, actually. I mean, we're beginning to get depressed about it. We've been doing it for a good five or six years, and I think we know how almost unanswerable the problem is. (RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 42.)

Dr. Graham W. Gibbs and Dr. Maurice Lachance in 1971 and 1972 conducted surveys in each of the five mines and mills in the Quebec chrysotile mining and milling industry. A total of 87 pairs of membrane filter samples and midget impinger samples were collected side by side at each location. The results did not satisfactorily provide a prediction of fibre counts from impinger counts, and the authors concluded that until satisfactory conversion factors were derived it would be preferable to base standards for at least mines and mills on particle counts for which at the time there existed a reasonable body of evidence. See Graham W. Gibbs and Maurice Lachance, "Dust-Fiber Relationships in the Quebec Chrysotile Industry," *Archives of Environmental Health* 28:2 (February 1974): 69-71. Dr. Kenny S. Crump suggested in his evidence that the correlation between fibre counts and dust counts is less the lower the exposure levels. See RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, pp. 17-19.

(iv) There is likely considerable inaccuracy or at least variability in the measurements that do exist. This adds to the unreliability of the exposure data from the cohort being studied. It also raises concerns about comparing the results of different cohort studies. Even when counted by a trained operator, some variability in the measurement of the same sample may occur;[72] this effect is doubtless compounded when the operator is not qualified, as had often been the case in the years covered by the historical studies. Improving technology and changing methodology of measurement over time represent a further source of variability.[73] For example, if one were to go back in time using today's technology and methodology to measure the same dust cloud, undoubtedly one could produce a higher figure now than was originally counted.[74]

(v) Even where measurement data exist there is no information on the size distributions of fibre concentrations. There is also no information on climatic conditions that prevailed in the various plants being studied (for example, whether conditions were dry or humid). These conditions may have affected fibre levels and fibre size distributions.

(vi) There are no measurements at the low end of the dose-response curve, which is the area of current and future concern. It is only in the last decade or so that industry exposures have been reduced to current levels. Accordingly, we have no measurements at or below 2 f/cc sufficiently far back in time to enable us to look at the actual effect of these levels. In order to attempt any assessment of the disease risk at such levels, we are therefore compelled to extrapolate beyond actual observation.[75]

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[71] RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, p. 60; RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 112; Gibbs and Lachance, "Dust-Fiber Relationships in the Quebec Chrysotile Industry"; RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 44-45; and Yehia Y. Hammad, John Diem, and Hans Weill, "Evaluation of Dust Exposure in Asbestos Cement Manufacturing Operations," *American Industrial Hygiene Association Journal* 40:6 (June 1979): 490-495.

[72] RCA Transcript, Evidence of Dr. Graham W. Gibbs, 13 November 1981, Volume no. 33, p. 39; Graham W. Gibbs et al., "A Summary of Asbestos Fibre Counting Experience in Seven Countries," *Annals of Occupational Hygiene* 20:4 (December 1977): 321-332; G.W. Gibbs, R.S. McCullough, and R.A. Tate, "A Canadian Asbestos Counting Trial," *Canadian Journal of Public Health* 70 (September/October 1979): 343-350; and RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, p. 119.

[73] RCA Transcript, Evidence of Dr. Gerald R. Chase and Dr. Harrison B. Rhodes, 14 August 1981, Volume no. 27, p. 76. See also, RCA Exhibit II-27, Tab 19, in RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18: Eric J. Chatfield, "Airborne Asbestos Fibres: A Summary of Some Measurement Problems," Sheridan Park, Ontario Research Foundation, 1981, p. 3. (Mimeographed.)

[74] RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, p. 18; and RCA Transcript, Evidence of Dr. Gerald R. Chase and Dr. Harrison B. Rhodes, 14 August 1981, Volume no. 27, p. 76.

(vii) There is a lack of necessary individual data to provide a suitable base to compare the effects of short, intermittent, high exposures with continuous but lower exposures. In other words, the exposure data that do exist do not distinguish which individuals received their dose at a constant level and which individuals received their dose in shorter but more intense bursts.[76]

Smoking Data — The availability of information on the smoking habits of the cohort is important at least in terms of the incidence of lung and laryngeal cancer, where smoking is a contributing factor.[77] Such information generally has to be gathered by a personal interview or questionnaire of employees, or if deceased, of relatives. Frequently such information either is not available or has not been gathered by the investigator. Where it is available, it is often incomplete and is in any event of questionable reliability, as people are not always frank as to their true smoking habits.[78]

(c) Mortality or Morbidity Ascertainment

Completeness of Follow-up — This criterion relates to whether the epidemiologist has been able to trace his entire study population; that is, to ascertain what has actually happened to each member of that population. If his ascertainment rate is low, then one need be concerned as to whether the persons not traced are more likely to have died of asbestos-related disease than those accounted for, in which case even the level of mortality indicated by the study would underestimate the true mortality. Of course, such a bias could also work the other way, and there is generally no reason to expect a relationship between ascertainment rate and cause of mortality (or morbidity).[79] However, the fact remains that with less than a 100% ascertainment rate, there is bound to be uncertainty as to the overall level of mortality and potential for bias in the results. Fortunately, virtually all of the main epidemiological studies have trace rates of 90% or above. The exception is the published study by Weill, Hughes, and Waggenspack of the New Orleans asbestos-cement workers, where the trace rate is only 75%.[80] (However, Dr. Hans Weill is in the course of updating this study and has achieved a much higher ascertainment rate.)[81]

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[75] Sackett, "A Review of Diagnostic Tests for Causation," p. 15 and Appendix B, p. 8.

[76] Enterline, "Pitfalls in Epidemiological Research: An Examination of the Asbestos Literature," p. 154; RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 62; and RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, p. 121. Dr. A.D. McDonald testified that ". . . epidemiologically we have absolutely no means for distinguishing between the short, sharp or the continuous, low [dose]. . . ."

[77] See the discussion of "the effect of smoking" in Chapter 5, Section F of this Report.

[78] See the Appendix to Chapter 7 of this Report.

[79] RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 68–70; and RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 65.

End-Point Ascertainment — This criterion refers to the accuracy of the diagnosis of the diseases identified in the cohort and the availability of death certificates for all deaths. If the disease is identified on the basis of a death certificate (which is the usual method for determining cause of death), the epidemiologist must consider whether it is reasonable to accept the information there stated or whether to use additional information (from autopsy reports, for example) to confirm or, if necessary, correct the diagnosis. We have been told that death certificates are of limited reliability in determining whether death was caused by an asbestos-related disease.[82] This was particularly true for those deaths years ago when diseases from asbestos exposure were not as well known to the ordinary practising physician and pathologist and when, as a result, the possibility of misdiagnosis in favour of other better-known diseases was considerable.[83] Asbestosis and mesothelioma are in any event open to misdiagnosis if only because they are not often seen by physicians in the general practice of medicine.

It is noteworthy that for the purposes of international diagnostic classification, mesothelioma of the pleura and the peritoneum were not even assumed to be malignant until the Eighth Revision of the International

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[80] Hans Weill, Janet Hughes, and Carmel Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," *American Review of Respiratory Disease* 120:2 (August 1979): 345-354.

[81] Letter from Dr. Hans Weill, Pulmonary Diseases Section, Department of Medicine, Tulane Medical Center, New Orleans, Louisiana to the Royal Commission on Asbestos, 11 April 1983.

[82] It appears that death certificates are apt to be particularly unreliable in the case of mesothelioma. Dr. Kotin has written that the likelihood of incorrectly diagnosing mesothelioma is so great that review of diagnoses of mesothelioma by panels of experts is recognized as indispensable throughout the world. He has also noted that even among the experts from the United States Mesothelioma Panel, there is at best agreement on the diagnosis in only 80% of the cases under review. See RCA Exhibit II-30, Tab 9, in RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A): Johns-Manville Canada Inc., Written submission to the Royal Commission on Asbestos, prepared by Paul Kotin, M.D., #18, 15 January 1981, p. 9. See also, Alison D. McDonald, "Mesothelioma Registries in Identifying Asbestos Hazards," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 444-445; RCA Transcript, Evidence of Dr. Alexander C. Ritchie, 14 July 1982, Volume no. 49, pp. 92-93; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 85-87. Dr. Ritchie testified that pathologically mesothelioma is still a difficult diagnosis. Dr. Weill gave evidence that for a long period of time there had been an underdiagnosis of mesothelioma. He went on to say that mesothelioma continues to be a difficult diagnosis and that even the mesothelioma panels now operating in Canada, the United States, and Great Britain have not greatly reduced inter-observer variability in the diagnosis of this tumour. Dr. Weill also pointed out that some authorities have suggested that there is now an over-diagnosis of mesothelioma.

[83] RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 85-87; and A.D. McDonald, "Mesothelioma Registries in Identifying Asbestos Hazards," pp. 441-443.

Classification of Diseases (ICD) in 1965 and were not automatically considered so until the Ninth Revision in 1975. Malignant neoplasm of the pleura first received a separate classification in the Eighth Revision, and peritoneal mesothelioma was not adequately delineated until the Ninth Revision.[84] Despite the increasing awareness of the effects of asbestos exposure, and despite increasing medical knowledge of asbestos-related disease, there remains serious inaccuracy in the certification of this cause of death. Dr. J. Corbett McDonald and Dr. Alison D. McDonald, who have extensively investigated the etiology of mesothelioma, have estimated that inaccuracy may be as high as 50%.[85] By way of example, Dr. A.D. McDonald cited the fact that in the province of Quebec, one-half of all mesothelioma cases registered through pathologists could not have been identified from death certificates.[86] Given this inaccuracy, and given the fact that the incidence of mesothelioma or indeed asbestosis in the general population is minimal, there is at least some justification to go behind the death certificate and search for the best available evidence as to the true cause of death.[87] However, supplementing death certificate information with other information and in effect basing the cause of death in the study population on information different from that available for the control population runs the risk of biasing any comparisons that are made.[88]

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[84] A.D. McDonald, "Mesothelioma Registries in Identifying Asbestos Hazards," pp. 444–445. In the Eighth Revision of the ICD, Code 163 is for "malignant neoplasms of other and unspecified respiratory organs." Code 163.0 is for "malignant neoplasms of pleura." In the Ninth Revision, Code 163 is for "malignant neoplasm of pleura" with the following subheadings: 163.0, parietal; 163.1, visceral; 163.8, other; and 163.9, pleura, unspecified. Pursuant to the Seventh Revision of the ICD, a number of pleural mesotheliomas were coded to 212, "benign neoplasms of the respiratory system," or, if the site of the tumour was not specified on the death certificate, to the classification for "benign neoplasms of the connective tissue" or "benign neoplasms of other and unspecified sites."

In the Eighth Revision, Code 158 is for malignant neoplasms of the peritoneum and retroperitoneum tissue. The only subheadings are Code 158.0 for retroperitoneal tissue and Code 158.9 for other parts. In the Ninth Revision, Codes 158 and 158.0 remained as in the Eighth Revision. However, there was a further breakdown and Code 158.8 was added for "specified parts of the peritoneum," while Code 158.9 is used for the "peritoneum, unspecified." Most of the neoplasms coded to 158.9 are peritoneal mesotheliomas. See *Eighth Revision International Classification of Diseases, Adapted for Use in the United States*, Public Health Service Publication, no. 1693 (Washington, D.C.: U.S. Government Printing Office, 1968); *International Classification of Diseases*, 1975 [Ninth] Revision (Geneva: World Health Organization, 1977); and *Comparability of Mortality Statistics for the Seventh and Eighth Revisions of the International Classification of Diseases, United States*, Department of Health, Education and Welfare Publication, no. (HRA) 76–1340, Vital and Health Statistics Series 2, no. 66 (Rockville, Maryland: U.S. Department of Health, Education and Welfare, October 1975); and Telephone communication between Mrs. Marian Heid, Nosologist, U.S. National Cancer Institute, Bethesda, Maryland and Royal Commission on Asbestos Staff, 8 November 1983.

[85] J. Corbett McDonald and Alison D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," *Preventive Medicine* 6:3 (September 1977): 432–433.

[86] A.D. McDonald, "Mesothelioma Registries in Identifying Asbestos Hazards," pp. 444–445. Dr. J.C. McDonald and Dr. A.D. McDonald have suggested that Quebec pathologists in general have over-diagnosed mesothelioma. They indicated that only 37% of cases examined from Quebec were accepted by the Mesothelioma Panel of the Canadian Tumour Reference Centre as compared with about 60% from other regions. Overall, they have concluded that the true rate of recognized mesothelioma is probably generally somewhat higher (perhaps by 50% or more) than that obtained through pathologists. J.C. McDonald and A.D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," pp. 430–433. Dr. Nicholson testified that in his opinion approximately 30% of death certificates are incorrect in respect of mesothelioma. He suggested, for example, that cancer of the pancreas happens to be a common diagnosis for peritoneal mesothelioma on death certificates. RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 12–14.

Length of Follow-up — The period between the time of first exposure of the last entrants into the cohort and the date upon which the investigator has undertaken his study must be sufficiently long to ensure that the diseases of interest will have time to manifest themselves. Bearing in mind the latency period before the appearance of malignant tumours or clinical fibrosis, most of the studies have a minimum follow-up period of at least 15 years.[89] There are, however, exceptions and, for example, there is no minimum follow-up period in the study by Thomas et al. of asbestos-cement workers at Cardiff[90] and only a 10-year minimum follow-up period in the Berry and Newhouse study of the Ferodo plant.[91]

(d) Statistical Analysis

Suitability of External Comparison Group — Ideally, the epidemiologist would want to construct a comparison population or control group out of the very type of cohort being studied. If he was studying insulation workers exposed to asbestos, his optimal control group would be insulation workers not so exposed. However, the data to do this simply do not exist and accordingly most investigators are obliged to utilize local, province- or state-wide, or more usually, national population bases. When the national population provides the comparison group, there may be problems occasioned by the fact that the cohort reflects a mortality experience peculiar to the particular region in which the study was conducted or by reason of factors that are peculiar to, or characteristic of, working populations but are not evident in the general population.[92] On the other hand, if province, state, or other local population mortality data are used, they may be overly contaminated by the very cohort being studied.[93]

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[87] RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 12-13; and Selikoff, Hammond, and Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," p. 114.

[88] Enterline, "Pitfalls in Epidemiological Research: An Examination of the Asbestos Literature," p. 152.

[89] See Chapter 5, Section B of this Report for a discussion of the major studies.

[90] H.F. Thomas et al., "Further Follow-up Study of Workers from an Asbestos Cement Factory," *British Journal of Industrial Medicine* 39:3 (August 1982): 273-276.

[91] Berry and Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos."

[92] See the discussion of this issue in Enterline, "Pitfalls in Epidemiological Research: An Examination of the Asbestos Literature," p. 153.

Occupational groups tend to be healthier and thus exhibit lower mortality from all causes than the general population. This fact, the so-called "healthy worker effect," means that when general population mortality data are used for the purpose of external comparison, it may underestimate the magnitude of the health effect of the substance of interest.[94] However, both Dr. J. Corbett McDonald and Mr. Geoffrey Berry, in their evidence before this Commission, indicated that while the healthy worker effect indeed biases some occupational studies, it is less likely to be a significant factor in the cases of diseases of long latency which affect older workers, like cancer or fibrosis; and from their testimony it would seem that the effect disappears after 15 years.[95]

Internal Comparison — Some cohort studies provide a separate internal comparison of certain aspects of the study, usually by a case-control analysis. The basic methodology in a case-control analysis is discussed below. Such an analysis may be used in conjunction with a cohort study to facilitate an investigation of the relationship between smoking, asbestos exposure, and lung cancer or to explore the influence of fibre type on the incidence of mesothelioma in the cohort. To the extent that such internal comparisons are made, they are helpful in supplementing and confirming the findings of the main cohort study, thereby adding to its reliability. Dr. J. Corbett McDonald's studies in Quebec and Mr. Julian Peto's recent study at Rochdale, England, have made particularly effective use of this methodology.[96]

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[93] Dr. Dement has come under some criticism for his use of national mortality rates. Since the local counties in and around Charleston have a very high lung cancer rate — approximately 75% above the national average — had Dr. Dement compared with local rates, the incidence of excess disease would have been less. Dr. Dement rejected the use of local rates for comparison because of the effect that the plant being studied may have had on local lung cancer death rates and because of the likely increases in those rates due to the local shipyard industry. Even so, the rates for the contiguous counties would have increased expected lung cancer rates only by 15% and the state rate was close to the national rate. See Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Workers," pp. 879–880.

[94] Enterline, "Pitfalls in Epidemiological Research: An Examination of the Asbestos Literature," p. 153.

[95] RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 138; and RCA Transcript, Evidence of Mr. Geoffrey Berry, 19 June 1981, Volume no. 11, p. 69. See also, RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, p. 85; and Enterline, "Pitfalls in Epidemiological Research: An Examination of the Asbestos Literature," p. 153.

Time Since First Exposure — All authors are aware of the need to allow an appropriately long latency period in calculating the disease risks of asbestos, and all studies calculate disease risks beginning at least 10 years (and more often 15 or 20 years) from first exposure.

The Index of Dose — The conventional index of dose is exposure, and the general method of quantifying the dose of asbestos that a person has received is by a measure of cumulative exposure. In many studies dose and exposure are used interchangeably. In fact, the concentration of asbestos to which a person is exposed is not necessarily the same as the dose of asbestos that person actually inhales.[97] And while ideally we may wish to know the dose inhaled, what we actually measure is exposure. In this Report, while we too use the terms interchangeably, we do so in light of the above-stated qualification. The measure of cumulative exposure is arrived at simply by multiplying the concentration of asbestos to which a worker was exposed by the number of years of exposure. However, in the case of asbestos exposure, there may be disadvantages to this general method. If the exposure is accumulated over short periods of time at high concentrations as, for example, with a demolition or maintenance worker, its effect may be different than if the same overall exposure was accumulated over a long period of time at much lower concentrations as, for example, with a worker in fixed place industry.[98] A measure of cumulative exposure simply does not permit exploration of these differences. For example, it does not distinguish between exposures of 2 f/cc for 50 years; 5 f/cc for 20 years; 50 f/cc for 2 years; and 100 f/cc for 1 year. All four exposures give the same measure of cumulative exposure: 100 f/cc-yrs. Further, a measure of cumulative exposure does not enable the epidemiologist to determine whether earlier exposures are more important than later exposures in the incidence of asbestos disease. While certain theoretical models have been developed to account for the residence time of asbestos in the lung,[99] there is no practical way to determine the real importance of residence time, and the measure of cumulative exposure used in all cohort studies treats all exposures equally, no matter how and when they were received. One final problem with asbestos studies, as with all studies of chronic disease arising from exposure to hazardous substances, is the inability to separate out completely the effects of dose from the effects of duration of exposure.[100] Ideally, the investigator would wish to separate completely the acquisition of exposure from the measurement of its effect so that one does not contaminate the other. The classic situation involving radiation disease where this was indeed regrettably possible was the dropping of the atomic bomb at Hiroshima and Nagasaki.[101] However, the follow-up period and the dose period invariably run together in asbestos cohort studies, which may blur the true nature of the dose-response relationship.

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[96] J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75"; J.C. McDonald and Liddell, "Mortality in Canadian Miners and Millers Exposed to Chrysotile"; Julian Peto, "The Incidence of Pleural Mesothelioma in Chrysotile Asbestos Textile Workers," in ***Biological Effects of Mineral Fibres***, vol. 2, pp. 703-711; and Julian Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," in ***Biological Effects of Mineral Fibres***, vol. 2, pp. 829-836.

[97] Simply because a worker is exposed to a given concentration of asbestos does not necessarily mean that the individual inhales all of it.

[98] RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 41-42; RCA Transcript, Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41, p. 54; RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), p. 36; and RCA Transcript, Evidence of Mr. Richard A. Lemen, 8 July 1981, Volume no. 17, p. 40. See the discussion of this issue in Chapter 5, Section G of this Report.

Analysis — The methods of analysis in the studies we have considered are sophisticated and complex and a detailed treatment in the text of this Report is not essential to understanding the significance of the results. We recognize that the analytical methods employed in the various studies differ to some extent.[102] However, we are satisfied that these different methods do not invalidate the results. Where appropriate, we will comment when the choice of a particular method of analysis may have affected the interpretation of the results.

D.3 The Case-Control Study

The basic design of a case-control study is to define a group of individuals who have some specified disease (the cases) and a second group who do not have the disease (the controls). The cases and the controls are then compared in terms of a factor or factors of interest.[103] One particular type of study that commonly uses a case-control analysis is a study which examines the fibre content of lung tissue in mesothelioma victims in order to assess which fibre type was responsible for causing the tumour.[104]

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[99] Geoffrey Berry and Hilton C. Lewinsohn, "Dose-Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards, Part I. Morbidity," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 185-194. Mr. Berry, in his evidence before this Commission, suggested that the traditional way of measuring dose by cumulative exposure appears unrealistic because it does not take account of when the exposure took place. The various models developed in the paper that he co-authored with Dr. Lewinsohn weighted the exposures by the time that the asbestos dust remained in the lung, but assumed that there was elimination of the dust. Thus, exposure that took place, for example, 25 years ago was considered more important in terms of disease production under the Berry and Lewinsohn models than exposure which took place, say, 5 years ago. See RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, pp. 25-27, 65-66. Dr. Crump, in his evidence, suggested that the health effect of different time patterns of exposure is not understood very well at all. See RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, pp. 22-24.

[100] RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 38-40; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 31-32.

[101] RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 31-32.

[102] For example, see Chapter 5, Section B of this Report.

The case-control study is not considered to be as strong an analytical tool as the cohort study because it is more prone to selection bias in the control group.[105] Still, such analysis does provide useful evidence, particularly when it is done in conjunction with cohort analysis.

D.4 The Case-Series Study

Here an investigator simply reports a series of cases among a defined population group. No comparison group is provided. Dr. Sackett has suggested that case series are prone to over-interpretation and are best used to stimulate further investigation of the problem by other methods.[106] A very good illustration of the value of a case-series report is that by Wagner, Sleggs, and Marchand of mesothelioma victims in South Africa beginning in 1960.[107] This case-series report not only alerted the medical community to the association between asbestos and mesothelioma, but of course stimulated the investigation of that association elsewhere in the world and by other more rigorous methods.

E. The Human Experience: Lung Tissue Studies

Lung tissue studies concerning asbestos are of recent vintage, with most if not all documented work having been published in the last decade.[108] They are thought to provide important evidence on two questions: first, as to whether the victim had been exposed to asbestos; and second, as to the relative pathogenicity of asbestos fibres of different types and sizes.

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[103] Sackett, "A Review of Diagnostic Tests for Causation," p. 12 and Appendix B, p. 3.

[104] See, for example, J.S.P. Jones et al., "The Pathology and Mineral Content of Lungs in Cases of Mesothelioma in the United Kingdom in 1976," in *Biological Effects of Mineral Fibres*, vol. 1, pp. 187-199; Alison D. McDonald, J. Corbett McDonald, and Fred D. Pooley, "Mineral Fibre Content of Lung in Mesothelial Tumours in North America," *Annals of Occupational Hygiene* 26:1-4 (1982): 417-422; and Neil Rowlands, Graham W. Gibbs, and Alison D. McDonald, "Asbestos Fibres in the Lungs of Chrysotile Miners and Millers — A Preliminary Report," *Annals of Occupational Hygiene* 26:1-4 (1982): 411-415.

[105] Sackett, "A Review of Diagnostic Tests for Causation," p. 12 and Appendix B, p. 3.

[106] Ibid.

[107] J.C. Wagner, C.A. Sleggs, and P. Marchand, "Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province," *British Journal of Industrial Medicine* 17 (1960): 260-271.

The typical lung tissue study is a case-control analysis in which the asbestos fibre content of the lung tissue of the series of cases (persons who died of asbestos-related disease, usually mesothelioma) is compared with a matched series of controls, with the specimen analysis done by electron microscopy. The investigator seeks to draw conclusions as to the causal relationship between exposure and disease based on whether or not the fibre content of the lungs of the cases is or is not significantly elevated above that of the controls.

The principal reservation concerning lung tissue studies is the extent to which the tissue examined at death reflects actual exposure in life. When an examiner looks at autopsy material, he is looking at what is residual and not necessarily at what was originally deposited there and may have been removed during life. This creates the concern that the fibres which may have been there initially and caused the trouble have been removed before the lung tissue is examined.[109] The amount of asbestos fibre retained in the lung is likely underestimated in any event due to the difficulty encountered in its recovery from the lungs at autopsy, and this difficulty likely applies more to chrysotile than to the other fibres because of chrysotile's greater solubility and its apparent tendency to break down chemically and physically after a long residence time.[110]

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[108] Jones et al., "The Pathology and Mineral Content of Lungs in Cases of Mesothelioma in the United Kingdom in 1976"; A.D. McDonald, J.C. McDonald, and Pooley, "Mineral Fibre Content of Lung in Mesothelial Tumours in North America"; and Rowlands, Gibbs, and A.D. McDonald, "Asbestos Fibres in the Lungs of Chrysotile Miners and Millers — A Preliminary Report." See also, Fred D. Pooley, "Methods for Assessing Asbestos Fibres and Asbestos Bodies in Tissue by Electron Microscopy," in *Biological Effects of Asbestos*, eds. P. Bogovski et al., IARC Scientific Publications, no. 8 (Lyon, France: International Agency for Research on Cancer, 1973), pp. 50–53.

[109] RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 100; and RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 90.

[110] Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," p. 194.

F. Animal Studies: The Advantages and Disadvantages of Animal Experiments with Asbestos

Animal experiments have become an important aspect of the estimation of the risk to humans of cancer and other diseases;[111] and animal testing has been widely used to study the health effects of asbestos. Three different types of animal experiments are relevant for the study of asbestos-related disease: injection experiments, inhalation experiments, and ingestion experiments.

The underlying premise of animal experiments is that the effects of properly conducted animal tests are applicable to humans. Considerable assurance as to the general applicability of this premise is derived from the fact that compounds generally known to be carcinogenic in humans are also carcinogenic in animals (save arsenic and perhaps benzene) and from the fact that no demonstrably significant aspect of the natural occurrence, induction, and properties of cancer has been shown to differ fundamentally between humans and experimental animals.[112]

Having regard to these considerations, we accept the conventional wisdom of the medical and scientific community that animal experiments are useful as a qualitative indication of potential human health effects in relation to asbestos exposure. Their great advantage is that they can be done over shorter time with greater precision and control than can be achieved by epidemiological methods.[113] Animal studies enable the investigator to explore particular issues for which the data from human experience may not be available or may not be reliable. For example, they permit an investigation of the importance and effect of variations in fibre size, the break-up of fibres in lung tissues, and the effects of different fibre types under controlled conditions.

While we recognize the value and importance of the results of animal testing with asbestos, we are also mindful of their limitations. One such limitation is that the asbestos samples generally used in animal experiments to date have not been used commercially. For example, neither the superfine Canadian chrysotile nor the standard UICC (Union Internationale

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[111] Crump et al., "Fundamental Carcinogenic Processes and Their Implications for Low Dose Risk Assessment," p. 2973; and "The OSHA Cancer Policy," pp. 5060ff.

[112] "The OSHA Cancer Policy," pp. 5060-5067; and John M.G. Davis, "The Use of Animal Models for Studies on Asbestos Bioeffects," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 795-798.

[113] Ibid. RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 39-40, 143; RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, p. 60; and RCA Transcript, Evidence of Mr. Richard A. Lemen, 8 July 1981, Volume no. 17, p. 11.

Contre le Cancer) chrysotile, amosite, or crocidolite which are utilized in much of the experimental work are used in industry.[114] The fibres in these experimental samples are indeed usually much finer than their industrial counterparts, and in the result their use in animal experiments blurs the potential differences between the effects of fibre type and of fibre dimension. Of course, to the extent that the trend in modern asbestos industry is to utilize finer fibres, animal experiments using UICC samples become that much more relevant, a point that was made by Dr. E. Donald Acheson in his report to the U.K. Advisory Committee on Asbestos and in his evidence before us.[115]

The most precise of animal experiments, the intrapleural injection experiment, is also the most unrealistic in comparison with the human experience.[116] Humans breathe or ingest asbestos fibres. Injection experiments implant or inject dust containing asbestos onto the lung or pleura, thereby avoiding the need to travel to the site of interest and bypassing the defence mechanisms of the respiratory system. However unrealistic they may be in terms of how the fibres reach the pleura, intrapleural injection experiments still do allow the investigator to test directly the effects of asbestos once deposited in the pleural cavity. Inhalation experiments are more realistic than injection experiments in that they more closely simulate human exposure, but they are also harder to interpret since the penetration of dust through the airways and alveoli will vary with time and will differ with different samples of dust. For the results to be of value, the dust cloud must be uniform and accurately monitored. However, Dr. Davis testified that in most animal inhalation experiments, there is a relatively poor characterization of the dust cloud in terms of two possibly important variables: fibre size and fibre shape.[117]

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[114] RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 29, 76-77; John M.G. Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature* (Edinburgh: Institute of Occupational Medicine, December 1980), pp. 17-19; and RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 33.

[115] E. Donald Acheson and Martin J. Gardner, "The Ill Effects of Asbestos on Health," in U.K., Advisory Committee on Asbestos, *Asbestos — Volume 2: Final Report of the Advisory Committee* (Simpson Report), William J. Simpson, Chairman (London: Her Majesty's Stationery Office, 1979), paragraph 168, pp. 33-34; and RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 11. See also, J. Christopher Wagner et al., "The Effects of the Inhalation of Asbestos in Rats," *British Journal of Cancer* 29:3 (March 1974): 268.

[116] J. Christopher Wagner, Geoffrey Berry, and Victor Timbrell, "Mesotheliomata in Rats After Inoculation with Asbestos and Other Materials," *British Journal of Cancer* 28:2 (August 1973): 177; and Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, p. 11.

[117] Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, p. 11.

A frequent criticism of animal experimental studies is that due to inter-species differences, extrapolation to humans is not justified. While this may be an extreme view, it is important that the animal model for experimental pathologic studies should duplicate as closely as possible the known human pathologic conditions intended to be studied.[118] Most experimental animal studies of asbestos disease have used the rat because the biological mechanisms of importance as between the rat and the human are thought to be similar.[119] However, in at least one respect, the tissue reaction of rats to asbestos fibres is dissimilar to that of humans. Rats rarely, if ever, form asbestos bodies. At present, it is uncertain whether the inability of rats to form these bodies is a disadvantage to their use in animal experiments.[120]

In order to induce cancer in animals within their lifetime, the doses of asbestos to which animals are exposed in laboratory testing are extremely high, certainly much higher than those to which workers are likely to have been exposed in recent years.[121] This gives rise to another important limitation on the value of animal experiments with asbestos. They cannot at the present time give precise information on the quantitative risk of exposure in humans.[122] Accordingly, while animal experiments are useful in a qualitative sense to indicate the existence of a dose-response relationship, they are of dubious assistance in determining the shape of the dose-response curve in humans. Because animals are given high doses over short periods, quantitative comparison with, or quantitative extrapolation to, the human experience is of doubtful reliability.

Laboratory animals have a much shorter lifespan and therefore a much shorter period for tumour induction than humans. This shorter time period may mean that any potential health effects in humans arising from the residence time of fibres in the lungs or arising from the physical process by which fibres break up may not be discoverable from animal experiments. More generally, animal experiments with asbestos will not be able to cast any light on the long-term chemical effects of fibres if indeed such effects exist.[123] While the rat's shorter lifespan may in one sense detract from the value of animal experiments, it is very advantageous from another point of view. It allows identification of the various effects of a substance well before the results of epidemiological studies in humans are known. This makes animal experiments particularly important in assessing the disease potential of new substances or substitutes for currently used substances.[124] But even with a substance such as asbestos where there is already a body of epidemiological data, animal experiments may provide new information as to its health effects and, where appropriate, enable preventive action to be taken in advance of such information being made known from epidemiology.

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[118] Davis, "The Use of Animal Models for Studies on Asbestos Bioeffects," pp. 795-796.

[119] Ibid. See also, Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, pp. 17-19; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 134-135. Dr. Davis testified that the size of the air space of the alveoli is not too much different in the rat than in the human. He also indicated that the macrophages for a rat are very similar in size as for a human and that the rat will be inhaling from any dust cloud probably the same fraction of asbestos fibres compared with body size and weight as the human being.

[120] Davis, "The Use of Animal Models for Studies on Asbestos Bioeffects," p. 796.

[121] Crump et al., "Fundamental Carcinogenic Processes and Their Implications for Low Dose Risk Assessment," p. 2973; and "The OSHA Cancer Policy," pp. 5060ff.

[122] RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), p. 9.

G. *In Vitro* Studies

In vitro or cell culture studies have been used as an alternative to animal experiments to investigate the biologically relevant characteristics of asbestos. For example, several authors have demonstrated the dependence of toxicity on fibre length in *in vitro* systems. Others have demonstrated that durability or solubility of fibres may be an important factor and that chrysotile in particular can be broken down in organisms.[125]

The advantages of *in vitro* experiments, in addition to the considerable savings in time and cost that they occasion, are that they provide an opportunity to study events at the cellular level under controlled, quantifiable, and reproducible conditions. Their disadvantage is that they take place in isolation from the normal integrating mechanisms to which the cell is subjected in the whole organism. The extrapolation of the results of *in vitro* studies with asbestos to humans or even animals has been limited, and it would appear that they are best used to supplement the findings of epidemiology and animal experiments in relation to the pathogenesis of asbestos.[126]

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[123] RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 46; RCA Transcript, Evidence of Mr. Julian Peto, 30 July 1981, Volume no. 25(B), p. 136; Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, pp. 17-19; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 30-32. If the time for break-up of chrysotile is roughly the same in animals as in humans, then it would take place over most of the rat's lifetime, but a very short percentage of a human's lifetime.

[124] "The OSHA Cancer Policy," pp. 5060ff.; and Crump et al., "Fundamental Carcinogenic Processes and Their Implications for Low Dose Risk Assessment," p. 2973.

[125] E.G. Beck, "Experimental Pathology — *In Vitro* Studies — Related to Asbestos and Other Mineral Fibres," in *Biological Effects of Mineral Fibres*, vol. 1, pp. 385–400.

[126] Ibid., p. 385.

Chapter 5 The State of the Evidence on Major Health Questions

A. Introduction

In Sections B, C, and D of this chapter we address the question: What are the important determinants of the health hazards of asbestos? First we consider the use of asbestos in different types of industrial processes. We do so by reviewing the major cohort studies of asbestos workers. The findings of these studies appear to indicate that the health hazards of asbestos vary significantly between different industrial processes. We then consider the health effects of different fibre types. Here the epidemiological data appear to indicate that the amphiboles are more hazardous than chrysotile, particularly in relation to the production of mesothelioma. We then discuss what might account for these differences. The available data indicate that there is an association between fibre dimension and asbestos-related disease. Long, thin fibres, that is, fibres longer than 5 or perhaps 8 microns in length and thinner than 1.5 or perhaps 0.25 microns in diameter, appear to be particularly implicated in the pathogenicity of asbestos. And just as there is an association between fibre dimension and disease, there appears in turn to be an association between type of industrial process and fibre dimension. As well, there appears to be a certain association between fibre type and fibre dimension.

While we consider that the dimension of the fibres offers the main explanation for the health hazards arising from asbestos, we acknowledge that other factors — fibre chemistry and durability — may play a role. We also acknowledge that our conclusions must be couched in a degree of uncertainty. Just as there exists medical uncertainty as to the mechanisms by which the asbestos-related diseases are caused, so there remains uncertainty as to the biologically relevant characteristics of asbestos.

In Sections E, F, G, H, and I of this chapter we consider some specific health issues with policy implications. In Section E we examine the nature of the dose-response relationship for the asbestos-related diseases and conclude that a linear relationship satisfactorily describes the dose-response relationship for asbestosis, lung cancer, and mesothelioma. We also find that while the weight of the available data indicates that there is now a low level of occupational exposure at or below which the clinical manifestation of asbestosis is most unlikely, we find that no such conclusion is justified either for lung cancer or mesothelioma.

In Section F we consider the effect of smoking on the incidence of disease. We note that smoking cannot initiate either mesothelioma or asbestosis, although it may enhance the progression of the latter. In contrast, not only can smoking initiate lung cancer, but in combination smoking and asbestos appear to act synergistically in the production of the tumour.

In Section G we consider the health effects of peak exposures or short, intense doses of asbestos. We do so out of concern that exposure of this kind is characteristic of occupations that bring workers into contact with asbestos through demolition, building maintenance, and repair work. From the limited data available we conclude that short, peak exposures to asbestos may pose a special health risk because of the possibility that such exposures will overwhelm the lung's clearance mechanisms.

We then discuss in Section H whether there is a special health risk to children from asbestos exposure. While we do not suggest that the levels of asbestos exposure outside the workplace themselves present a health risk to children or to any other members of the general public, we do indicate that the time course of asbestos-related disease, particularly mesothelioma, means that the earlier in life asbestos exposure first takes place, the greater the risk of disease.

Finally, we consider in Section I whether there is any health risk from the ingestion of asbestos fibres either through eating or drinking. This issue has raised public concern especially because of the presence of asbestos in many municipal water systems. We find that it is most unlikely that ingested fibres penetrate the gastrointestinal tract. In addition, there is a great deal of evidence that asbestos fibres found in water systems are very short and hence are not to be considered hazardous. (See Chapter 11 of this Report.) We conclude that the evidence fails to indicate any health risk from the ingestion of asbestos fibres.

B. The Effect of Industrial Process

B.1 Asbestos Mining

(a) Quebec

In 1966, a research team at McGill University headed by Dr. J. Corbett McDonald began investigating the health effects of a large group of chrysotile miners and millers working in the Eastern Townships of Quebec.[1] Dr. J.C. McDonald and his colleagues have reported the results of their findings several times over the years and have continued to update their data and improve their methodology.[2] The most recent report of their findings was published in 1980 and was based upon a cohort of 11,379 workers (10,939 men and 440 women) born between 1891 and 1920 who had worked for at least one month in the mines and mills of Asbestos and Thetford Mines, Quebec, and who had been followed to the end of 1975.[3] It is generally conceded that this report represents the most comprehensive and reliable epidemiological study of the mining and milling of chrysotile asbestos.[4] There is considerable exposure information. The authors identified 5,783 different jobs through detailed work histories. Individual cumulative dust estimates were made on the basis of over 4,000 midget impinger dust counts made personally by the same industrial hygienist in the years 1949–1966.[5] However, it must be observed that there were no measurements made in the open pit, only a limited number of measurements in the underground mines, and virtually no actual measurements of any kind in the pre-1949 period.[6] For the period prior to 1949, estimates had to be made on the basis of comparisons with more recent conditions following interviews with long-service employees.[7] Moreover, those measurements made after 1949 were of dust particles rather than asbestos fibres. While Dr. J.C. McDonald and his associates have recently been attempting to convert the dust particle measurements to fibre measurements, he frankly conceded in his testimony before this Commission that the results have been far from satisfactory.[8]

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[1] Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 10.

[2] J. Corbett McDonald et al., "Mortality in the Chrysotile Asbestos Mines and Mills of Quebec," *Archives of Environmental Health* 22 (June 1971): 677–686; J. Corbett McDonald et al., "The Health of Chrysotile Asbestos Mine and Mill Workers of Quebec," *Archives of Environmental Health* 28:2 (February 1974): 61–68; J. Corbett McDonald and F. Douglas K. Liddell, "Mortality in Canadian Miners and Millers Exposed to Chrysotile," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 1–9; and J. Corbett McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910–75," *British Journal of Industrial Medicine* 37 (1980): 11–24. Dr. J.C. McDonald and his colleagues have studied the Quebec miners and millers at four points in time: 1966, 1969, 1973, and 1975. See RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 10. For a detailed discussion of the methods of cohort analysis used by Dr. J.C. McDonald, see F.D.K. Liddell, J.C. McDonald, and D.C. Thomas, "Methods of Cohort Analysis: Appraisal by Application to Asbestos Mining," *Journal of the Royal Statistical Society* 140, Part 4 (1977): 469–491.

[3] J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910–75." In this latest report, Dr. J.C. McDonald and his colleagues used two main methods of analysis: a man-years approach (*a priori* analysis) and a case-controls approach (*a posteriori* analysis). RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 6.

[4] Michael Alavanja, Christine New, and Judy Parsells, "An Assessment of the Epidemiological Literature Related to Worker Exposure to Chrysotile in Mines and Mills," NIOSH internal document prepared for the U.S. Mine Safety and Health Administration, 1981. (Mimeographed.); RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 103; RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, p. 85; and RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 62.

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[5] J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910–75," p. 13. Dr. Graham W. Gibbs testified that he collected the data which Dr. Maurice Lachance had available for the mining industry for each year and by work area. He then looked at the concentrations and the pattern of dustiness for each work area over a period of time. Dr. Gibbs then took each of the jobs that each of the people in the industry had and looked at which work areas those jobs would have been associated with. He then assigned, for those years to those jobs, the dust concentrations associated with those particular work areas. See RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 29–30.

[6] J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910–75," p. 13; RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 5; and RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, p. 77.

[7] RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 29–31. Dr. Gibbs testified that the general view of long-term employees and of management was that dust conditions in the early 1950s reflected fairly well dust concentrations that existed almost back at the turn of the century. Accordingly, Dr. Gibbs and Dr. Lachance utilized the concentrations in the early 1950s to extrapolate backwards, modified somewhat by the information available from the employee and management groups. Dr. Gibbs is of the view that the raw dust data from the Quebec mines are probably far better than the data found for most epidemiological studies in the sense that although there are not measurements for every day and every year at all locations, there are essentially systematic measurements made by the same individual over a long period of time. See RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 58.

By the end of 1975, 4,463 men and 84 women (a total of 4,547 workers) had died out of the cohort of 11,379 workers (10,939 men and 440 women). There was a net excess of 33.9 deaths at Asbestos and 208.8 at Thetford Mines, yielding overall standard mortality ratios (SMRs) of 102 and 110 respectively. Approximately 10% of the cohort was not traced, but most were apparently workers with short service.[9]

The data, analyzed by different methods, gave results consistent with one another and with previous analysis. For total mortality, and for mortality from pneumoconiosis (which would be mostly asbestosis) and lung cancer, there were quite consistent trends to higher SMRs the greater the dust exposure.[10] The pattern for gastrointestinal cancer was uneven: there was substantial excess mortality from cancer of the upper gastrointestinal tract in the workers most heavily exposed (at Thetford Mines), but otherwise no excess, prompting the authors to suggest that some other causal factor was operative.[11]

There are at least two noteworthy findings of this study. First, among the 1,904 men with at least 20 years' employment in the lower dust categories, with an average yearly exposure of 6.6 million particles per cubic foot (mppcf) [or perhaps about 20 fibres per cubic centimetre (f/cc)], excess mortality, although not absent, was not considered to be statistically significant except for asbestosis.[12] The inability of such a large epidemiological study to demonstrate an increased risk for cancer at exposure levels far in excess of present levels suggests that any risk in chrysotile mining at such levels is very small. Indeed, the authors speculated in their published study that the dust concentrations in the Quebec mines and mills in the early 1950s carried a lung cancer risk equivalent to heavy smoking, whereas at more recent concentrations of around 1 mppcf the order of risk may approximate to less than one cigarette per day.[13] It may be that the cancer risk has been underestimated to some extent in this cohort, in part because the authors used provincial mortality data for their comparisons whereas the rural mining area of Quebec has a lung cancer mortality rate which is two-thirds of the provincial rate.[14] Still, it is evident that the overall disease incidence among these chrysotile miners and millers is relatively low save in the long-term and most heavily exposed employees.

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[8] J. Corbett McDonald, Graham W. Gibbs, and F. Douglas K. Liddell, "Chrysotile Fibre Concentration and Lung Cancer Mortality: A Preliminary Report," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 811-817. The authors used a case-control analysis of 244 deaths from lung cancer and 244 surviving controls matched for date of birth, mining area, and smoking habit. All measurements ever made in the industry using membrane filters or midget impingers were compiled although there was little overlap. These measurements yielded an average conversion ratio of 1 mppcf equals 3.14 f/cc. In the article, the authors suggested that, in the absence of evidence to the contrary, this average conversion ratio could usefully be applied to other results in the chrysotile mining industry. However, in his oral testimony before this Commission, Dr. Gibbs indicated that he was unhappy with this conclusion and would have preferred to have restricted the conversion factor of 3.14 as applicable only to the precise job distribution in the case-control study from which the figure came. See RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 66. In his own testimony, Dr. J.C. McDonald conceded that his group's attempt to convert dust particle measurements to fibre measurements is ". . . more or less fizzling out, actually. I mean, we're beginning to get depressed about it. We've been doing it for a good five or six years, and I think we know how almost unanswerable the problem is." See RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 42.

[9] J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," p. 11. The authors' mortality analyses focused on the 3,291 male deaths occurring 20 or more years from first employment and in the period 1951-1975. The figure 3,291 is used in both Tables 5.1 and 5.18 in this chapter.

[10] Ibid., p. 17.

[11] Ibid., pp. 22-23.

The second important finding of this study was that among the 4,547 deaths in this cohort, there were only 11 mesothelioma deaths, all of them pleural, 10 men and one woman.[15] Subsequent investigation by Dr. Alison D. McDonald and consideration of cases among those who did not qualify for inclusion in the cohort have increased the number to 21, 5 of whom also worked at a nearby factory which used both crocidolite and chrysotile.[16] Table 5.1 shows the standard mortality ratios for various causes of death in relation to duration of service for Dr. J.C. McDonald's cohort.

A smaller cohort of the Quebec miners has also been studied by Dr. William J. Nicholson and his colleagues at the Mount Sinai School of Medicine in New York.[17] They examined all men employed during 1961 at Thetford Mines with at least 20 years' seniority. The total cohort thus consisted of 544 persons, of whom 178 were dead by the end of 1977. The

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[12] Ibid., pp. 11, 23. In his evidence, Dr. J.C. McDonald emphasized that this finding did not indicate that there is no risk at such exposure. Rather, his conclusion was that there is a risk but that it cannot be demonstrated with statistical significance. See RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 23.

[13] J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," p. 21.

[14] RCA Transcript, Evidence of Dr. William J. Nicholson, 30 June 1981, Volume no. 15, p. 40; and Alavanja, New, and Parsells, "An Assessment of the Epidemiological Literature Related to Worker Exposure to Chrysotile in Mines and Mills," p. 12.

[15] J.C. McDonald and Liddell, "Mortality in Canadian Miners and Millers Exposed to Chrysotile," pp. 6-7; and J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," p. 11.

[16] Alison D. McDonald, "Malignant Mesothelioma in Quebec," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 673-680.

[17] William J. Nicholson et al., "Long-Term Mortality Experience of Chrysotile Miners and Millers in Thetford Mines, Quebec," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 11-21.

Table 5.1*

Deaths, by Cause, in Relation to Duration of Service

Cause of Death	Length of Gross Service (Years)									
	Very Short (<1)		Short (1<5)		Medium (5<20)		Long (>20)		Complete Cohort	
	O**	SMR***	O	SMR	O	SMR	O	SMR	O	SMR
All causes	885	1.07	629	1.09	679	1.15	1,098	1.07	3,291	1.09
Pneumoconiosis	1	1.15	3	5.00	2	3.39	36	34.62	42	13.56
Malignant neoplasms										
Lung	47	0.97	29	0.83	50	1.37	104	1.61	230	1.25
Esophagus and stomach	37	1.30	25	1.27	18	0.91	50	1.47	130	1.27
Colon and rectum	22	0.78	13	0.67	23	1.16	21	0.62	79	0.78
Other abdominal	20	1.08	12	0.92	14	1.04	21	0.90	67	0.98
Larynx	6	1.48	5	1.75	1	0.34	4	0.78	16	1.07
Other	67	1.12	43	1.04	48	1.13	79	1.08	237	1.09
Heart disease	370	1.06	251	1.02	287	1.15	424	0.97	1,332	1.04
Respiratory tuberculosis	7	0.62	7	0.89	21	2.68	22	1.56	57	1.39
Other respiratory	29	0.66	46	1.52	22	0.71	59	1.12	156	0.99
Cerebrovascular	62	0.95	49	1.12	50	1.13	82	1.11	243	1.07
Accidents	52	1.36	38	1.32	37	1.18	56	0.96	183	1.17
All other known causes	130	1.03	94	1.07	94	1.05	132	0.86	450	0.98
Cause not known	35	–	14	–	12	–	8	–	69	–

Notes: *[For all tables in this chapter, "O" or "Obs" mean observed deaths; "E" or "Exp" mean expected deaths; and "SMR" means standard mortality ratio.]

**Columns headed O give the numbers of deaths of men, 20 years or more after first employment, occurring during 1951-1975.

***Figures under headings SMR are ratios of deaths observed to those expected on the basis of male mortality in Quebec.

SOURCE: J. Corbett McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," *British Journal of Industrial Medicine* 37 (1980): 17 (Table 6).

SMRs for deaths from all causes and for lung cancer were 111 and 252 respectively. There was only one mesothelioma in the cohort and no excess of gastrointestinal cancer.[18] The cohort studied by Dr. Nicholson was heavily exposed and his results appear to be quite compatible with those of Dr. J.C. McDonald's most heavily exposed group for which the SMRs for death from all causes and for lung cancer were 130 and 225 respectively.[19]

(b) Italy

The disease incidence in chrysotile mining has also been investigated by Rubino et al. who examined the health experience of a group of chrysotile miners at the Balangero Mine in Northern Italy.[20] The cohort studied was quite small, consisting of 952 miners who had worked at least one month between 1930 and 1965 (and who were known to be alive in 1946). There were 332 deaths, giving an SMR for deaths from all causes of 155. Significant increases over expected were found, *inter alia*, for laryngeal cancer and non-malignant respiratory diseases, but not for lung cancer, where there were only 11 deaths and an SMR of 106. There was only one death from mesothelioma (pleural) which was included in the lung cancer calculation. However, the risk of asbestos-related disease may be underestimated in this study since mortality was examined as of the end of 1975, leaving a latency period of less than 20 years for individuals initially employed after 1955.[21] Dr. J.C. McDonald noted that the findings among the Italian miners were very similar to his own.[22]

(c) Australia

There has been at least one cohort study of crocidolite mining, that by Hobbs et al. who recently investigated a group of Western Australian crocidolite miners.[23] The study was based upon 6,200 male employees of the principal mining company in the region, followed to the end of 1977. No minimum interval from first employment was defined for this study, and accordingly, for many in the cohort the follow-up period was quite short. The SMR for death from all causes was only 90, perhaps in part attributable to a healthy worker effect. (See Chapter 4, Section D.2 of this Report.) However, the SMR for lung cancer was 157 (60 deaths versus 38.24 expected) and for pneumoconiosis, 1,045 (14 against 1.34 expected).[24] There were 26 cases of mesothelioma, all pleural, with a median interval from first exposure of approximately 20 years.[25] The incidence of mesothelioma was strongly related both to the duration and intensity of the crocidolite exposure.

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[18] Ibid., pp. 11-16. In 130 cases, the authors used clinical, surgical, or pathological data to supplement the information on the death certificates as to cause of death.

[19] See J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," Table 8, p. 19; and RCA Transcript, Evidence of Dr. William J. Nicholson, 30 June 1981, Volume no. 15, pp. 75-77. It is to be noted that Dr. Nicholson et al. studied only employees at Thetford Mines whereas Dr. J.C. McDonald et al. studied employees at both Asbestos and Thetford Mines. Overall, those employed at Asbestos had a lower mortality and disease experience than those employed at Thetford Mines.

[20] G.F. Rubino et al., "Mortality of Chrysotile Asbestos Workers at the Balangero Mine, Northern Italy," *British Journal of Industrial Medicine* 36 (1979): 187-194. The authors noted that the death certified as being caused by pleural mesothelioma occurred 35 years after starting employment in a worker with 33 years' exposure. Since there was no histological confirmation of the diagnosis, the authors accepted the diagnosis only with reservation.

[21] Alavanja, New, and Parsells, "An Assessment of the Epidemiological Literature Related to Worker Exposure to Chrysotile in Mines and Mills."

[22] J.C. McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," p. 22.

(d) South Africa

Within South Africa, crocidolite, amosite, and chrysotile have all been mined for many years. According to the final report of the United Kingdom Advisory Committee on Asbestos (the Simpson Report), the crocidolite mined in South Africa is of two types: one with extremely fine fibres which is mined in the northern part of the Cape province and the other, a more coarse variety mined in the Transvaal.[26] The amosite mines are found close to the crocidolite mines in the Transvaal, and the chrysotile mines are found in the south-east near the Swaziland border.

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[23] M.S.T. Hobbs et al., "The Incidence of Pneumoconiosis, Mesothelioma and Other Respiratory Cancer in Men Engaged in Mining and Milling Crocidolite in Western Australia," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 615–625.

[24] Ibid., p. 621. The authors have noted that the mortality ratio for all causes of death in the cohort, excluding both respiratory disease and accidents, is 0.80. Thus, there is a twofold difference between the mortality ratio for respiratory cancer and those for other causes of death.

[25] Ibid., pp. 618–620. The 26 cases of mesothelioma included cases diagnosed to July 1, 1979. The authors' mortality study, which indicated 17 mesothelioma deaths, was restricted to deaths before January 1, 1978. Mr. Julian Peto has noted the absence of peritoneal mesothelioma among the Western Australian crocidolite miners, which is in contrast to the other data which suggest that the risk of peritoneal mesothelioma is substantial after crocidolite exposure. See Julian Peto, "Discussion Summary," in *Biological Effects of Mineral Fibres*, vol. 2, p. 731. In their update on "The Ill Effects of Asbestos on Health" (which was prepared for the U.K. Advisory Committee on Asbestos), Dr. E. Donald Acheson and Dr. Martin J. Gardner have noted, on the basis of a personal communication from M.S.T. Hobbs, that as of 1983 there were 64 cases of mesothelioma among the Western Australian crocidolite miners: 60 pleural and 4 peritoneal. See E. Donald Acheson and Martin J. Gardner, *Asbestos: The Control Limit for Asbestos*, prepared for the U.K. Health and Safety Commission (London: Her Majesty's Stationery Office, 1983), paragraph 24, p. 4.

[26] E. Donald Acheson and Martin J. Gardner, "The Ill Effects of Asbestos on Health," in U.K., Advisory Committee on Asbestos, *Asbestos — Volume 2: Final Report of the Advisory Committee* (Simpson Report), William J. Simpson, Chairman (London: Her Majesty's Stationery Office, 1979), paragraph 130, p. 28.

Despite the extensive asbestos mining that has taken place in South Africa, there has been no systematic epidemiological study of the workers at risk so that case reports of disease must be considered cautiously. Nonetheless, it is noteworthy that according to Professor Ian Webster, of the 235 cases of mesothelioma reported in South African miners between 1956 and 1982, almost all were exposed to Cape crocidolite, a few to Transvaal amosite, and an occasional one to Transvaal crocidolite. No cases of mesothelioma were reported in relation to the mining of chrysotile but the chrysotile mining operation in South Africa is relatively small.[27]

(e) Asbestos-Contaminated Mines

In addition to asbestos deposits, asbestiform fibre concentrations are often found in other mineral deposits. For example, Mr. Richard A. Lemen of the United States National Institute for Occupational Safety and Health (NIOSH) testified before the Commission that data collected by the Institute indicated there were a number of mining operations in the United States in which at least measurable amounts of asbestos-like fibres were found.[28] Two examples were the Homestake Gold Mine located at Lead, South Dakota, where miners were occupationally exposed to fibrous cummingtonite-grunerite, that is, amosite; and the talc mines in New York State which are contaminated with deposits of tremolite.

The gold mine in South Dakota has now been subjected to three separate cohort studies.[29] An initial investigation by Gillam et al., published by NIOSH in 1976, focused on those 440 workers who by 1960 had completed at least 5 years of underground mining and found that there was excess mortality due to malignant and non-malignant respiratory disease particularly in the period 20 or more years from first exposure. The authors of the study concluded that neither silica exposure nor cigarette smoking could account for the observed increased lung cancer risk among these hard rock gold miners and accordingly attributed it to the exposure to the cummingtonite-grunerite.[30] Yet, a subsequent investigation of the same mine but of a differently constituted cohort by Dr. J.C. McDonald et al. could find no excess risk from lung cancer or any other malignancy.[31] Dr. J.C. McDonald conducted a mortality study of 1,358 workers who by 1973 had worked at least 21 years with the mining company. Almost all had spent most of their working lives underground. There was an increase in non-malignant respiratory mortality which appeared characteristic of hard rock mining with a severe silicotic risk.

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[27] Letter from Professor Ian Webster, National Centre for Occupational Health, Department of Health, Welfare and Pensions, Republic of South Africa to the Royal Commission on Asbestos, 14 July 1983. The findings of the Asbestos Tumour Reference Panel in South Africa list a total of 1,120 cases of mesothelioma between the years 1956 and 1982. Of this figure, 235 cases were attributed to exposure in mining and 178 cases to environmental exposure. In 453 of the cases, the source of exposure was either unknown or not available.

[28] RCA Transcript, Evidence of Mr. Richard A. Lemen, 8 July 1981, Volume no. 17, p. 63.

[29] J. Dean Gillam et al., "Mortality Patterns Among Hard Rock Gold Miners Exposed to an Asbestiform Mineral" (NIOSH Study), *Annals of the New York Academy of Sciences* 271 (28 May 1976): 336–344; J. Corbett McDonald et al., "Mortality After Long Exposure to Cummingtonite-Grunerite," *American Review of Respiratory Disease* 118:2 (August 1978): 271–277; and Samuel D. Kaplan and William R. Gaffey, "Miners Exposed to Amphibole Mineral: A Retrospective Cohort Mortality Study" (SRI Study), Cincinnati, Ohio, U.S. Department of Health and Human Services, June 1981. (Mimeographed.)

In light of the conflicting results of these two studies, NIOSH commissioned yet a further and more comprehensive study of the same mine by the Stanford Research Institute (SRI).[32] In this investigation, the study cohort consisted of 3,144 men employed underground in the mine for one year or more between 1940 and 1964 and followed to the end of 1977, by which time 827 men had died. The report of the SRI investigation, which was released in draft during the course of our own inquiry, strikingly confirmed the findings of Dr. J.C. McDonald and found no excess risk of lung cancer or indeed any other cause of mortality that could be associated with the grunerite exposure.[33] The authors of the report concluded that members of the cohort were at high risk of death from accidents and from tuberculosis and non-malignant respiratory disease related to silica exposure. The SRI authors also commented on the different results produced by the two earlier studies and observed that the study by Gillam et al. overestimated the lung cancer risk due to the lack of up-to-date mortality data for their analysis.[34]

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[30] Gillam et al., "Mortality Patterns Among Hard Rock Gold Miners Exposed to an Asbestiform Mineral," pp. 342–343.

[31] J.C. McDonald et al., "Mortality After Long Exposure to Cummingtonite-Grunerite," pp. 275–276. The authors of this study have suggested that the conclusions of Gillam et al. were weakened by the observation that the odds ratio for death from respiratory malignancies was greater within 20 years of first employment than after it. On the other hand, Mr. Richard A. Lemen, a co-author of the NIOSH study by Gillam et al., suggested in his testimony that Dr. J.C. McDonald and his colleagues were looking at a survivor population and, therefore, may have underestimated the risk. See RCA Transcript, Evidence of Mr. Richard A. Lemen, 8 July 1981, Volume no. 17, pp. 141–144. Dr. John M. Dement made the same point in his testimony. See RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, pp. 50–51.

[32] Kaplan and Gaffey, "Miners Exposed to Amphibole Mineral: A Retrospective Cohort Mortality Study."

[33] Ibid.

[34] Ibid. To calculate their expected numbers Gillam et al. used mortality rates for the general population only through the year 1967. The cohort was followed between the years 1960 and 1973, which means that for nearly one-half the person-years of follow-up, up-to-date general population mortality rates were not available and the earlier 1967 rates had to be used. The authors of the SRI study have suggested that for most causes of death this would have had little impact but that lung cancer mortality rates for males were increasing rapidly during that period of time. Thus, the expected number of deaths for lung cancer was unavoidably underestimated in the study by Gillam et al., which means that the corresponding SMR for lung cancer was unavoidably overestimated.

It is noteworthy that the cummingtonite-grunerite fibres found at this mine, while similar to amosite fibres in chemical constitution, were generally very short. Most were less than 5 microns in length with a median fibre length of 1.1 microns, a factor which, as we shall discuss, is consistent with the finding that these fibres played no role in the disease incidence at this mine.[35]

As in the United States, there are in Ontario several mining operations with low levels of asbestiform fibre concentrations. For example, chrysotile can be found in the talc and magnesite deposits in the Kirkland Lake - Timmins area.[36] The Ministry of Labour has recently taken measurements of the asbestos levels in some non-asbestos mines in Ontario and found that they are well below the current occupational control limit for chrysotile asbestos of 1 f/cc.[37]

B.2 Asbestos Manufacturing — Friction Materials

(a) Ferodo Plant, Derbyshire, England

Mr. Geoffrey Berry and Dr. Muriel L. Newhouse have recently conducted a mortality study of workers manufacturing friction materials at the Ferodo plant in Derbyshire, England. Mr. Berry reported the preliminary results of this study during his testimony before this Commission, and the authors' final report was published following the conclusion of our hearings.[38]

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[35] John M. Dement, Ralph D. Zumwalde, and Kenneth M. Wallingford, "Discussion Paper: Asbestos Fiber Exposures in a Hard Rock Gold Mine," *Annals of the New York Academy of Sciences* 271 (28 May 1976): 345-352.

[36] Ulrich Kretschmar and Dianne Kretschmar, *Talc, Magnesite, and Asbestos Deposits in the Kirkland Lake - Timmins Area, Districts of Timiskaming and Cochrane*, Ontario Geological Survey Open File Report 5391 (Toronto: Ontario Ministry of Natural Resources, 1982).

[37] Letter from Mr. Hugh M. Nelson, Special Assistant, Industrial Hygiene, Occupational Health and Safety Division, Ontario Ministry of Labour to the Royal Commission on Asbestos, 14 September 1981.

[38] The preliminary results were presented at the Fifth International Symposium on Inhaled Particles, conducted by the British Occupational Hygiene Society at Cardiff, Wales, in September 1980; and were subsequently published as: Muriel L. Newhouse, Geoffrey Berry, and Joseph W. Skidmore, "A Mortality Study of Workers Manufacturing Friction Materials with Chrysotile Asbestos," *Annals of Occupational Hygiene* 26:1-4 (1982): 899-909. For the final report, see Geoffrey Berry and Muriel L. Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," *British Journal of Industrial Medicine* 40:1 (February 1983): 1-7. The dust conditions in the plant have been separately discussed in J.W. Skidmore and B.L. Dufficy, "Environmental History of a Factory Producing Friction Material," *British Journal of Industrial Medicine* 40:1 (February 1983): 8-12.

The study was based on a cohort of 13,460 workers employed from 1942 onwards and traced to the end of 1979, by which time 1,986 had died.[39] Chrysotile was the only type of asbestos used in this plant except for two well-defined periods prior to 1949 when crocidolite was used. Regular measurements were introduced in the plant only in 1967 and fibre concentrations in earlier years were estimated by attempting to reproduce earlier working conditions. Given the passing of the 1931 Asbestos Industry Regulations and ventilation improvements in the plant, it was estimated that all the jobs within the plant except grinding and fibre preparation had exposures under 5 f/cc. By the 1950s, all exposures in the plant were estimated to be below 5 f/cc; and by 1969, pursuant to the then new regulations, exposures were reduced to less than 2 f/cc.

Table 5.2 indicates observed and expected mortality among men and women in the cohort after 10 years from first exposure to asbestos. Apart from 10 pleural mesotheliomas (and one further since the mortality analysis was completed), there was no evidence of any excess mortality among these workers either from all causes or from any particular cause including lung cancer (although it should be observed that the minimum period of follow-up was only 10 years).[40] And while a considerable number of the workers were employed for a short period of time, even those with the longest service, 20 or more years, and who were thus more heavily exposed, demonstrated no excess disease.[41] (Table 5.3 shows observed and expected mortality among those workers who had been employed at least 10 years.) As the authors commented, however, a noteworthy aspect of the study was the low exposures experienced by the workforce.[42] This occurred in part because of the number of short-term employees but was also a consequence of environmental control in the factory over an extended period of time. As a result, it is estimated that only 5% of the men employed after 1941 had a cumulative exposure of 100 fibres per cubic centimetre-years (f/cc-yrs). At 100 f/cc-yrs, Dr. J.C. McDonald estimated that the SMR for lung cancer

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[39] A further 187 deaths occurred in this cohort in the 18-month period commencing January 1, 1980, yielding a total of 2,173 deaths. Of these additional 187 deaths, 13 were due to lung cancer, and one man was certified as dying of a pleural mesothelioma. See Berry and Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," p. 3.

[40] Berry and Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," pp. 1-3.

[41] Ibid., p. 3. In September 1983, as we were completing our Report, Dr. Murray M. Finkelstein and Mr. Robert A. Kusiak released in draft the results of a mortality study they had conducted of former employees of the Bendix Automotive Corporation located at Windsor, Ontario. These employees were potentially exposed to chrysotile asbestos. The authors noted that the results of their study are in line with those at Ferodo and concluded that workers exposed to asbestos in friction materials operations face health risks which are at worst marginal. See Murray M. Finkelstein and Robert A. Kusiak, "A Study of Mortality Among Employees of the Bendix Automotive Corporation, Windsor, Ontario," Toronto, Ontario Ministry of Labour, September 1983. (Mimeographed.)

[42] Berry and Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," p. 6.

Table 5.2

Observed and Expected Mortality After 10 Years from First Exposure

Cause of Death	Number/Subject-Years			
	Men 7,474/104,193		Women 3,708/58,816	
	Obs	Exp	Obs	Exp
All causes	1,339	1,361.8	299	328.0
Lung and pleural cancer	151 (8)*	139.5	8 (2)	11.3
Gastrointestinal cancer	103	107.2	29	27.4
Other cancers	77	87.7	51	60.0
Other causes	1,008	1,027.4	211	229.3

Note: *Number of pleural mesotheliomas included in parentheses.

SOURCE: Geoffrey Berry and Muriel L. Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," *British Journal of Industrial Medicine* 40:1 (February 1983): 3 (Table 7).

Table 5.3

Observed and Expected Mortality After Completing 10 Years' Employment

	Men				**Women**			
Follow-up After 10 Years' Exposure (Years)	**0-10**		**>10**		**0-10**		**>10**	
Number/Subject-Years	**2,484/21,860**		**1,808/19,025**		**627/5,578**		**457/6,377**	
Cause of Death	**Obs**	**Exp**	**Obs**	**Exp**	**Obs**	**Exp**	**Obs**	**Exp**
All causes	185	195.7	432	450.8	14	21.3	76	66.5
Lung and pleural cancer	23	21.3	58 (7)	47.4	0	0.7	2 (1)	2.2
Gastrointestinal cancer	23	16.3	25	35.8	0	1.8	8	5.7
Other cancers	7	12.6	21	28.2	3	4.5	14	10.7
Other causes	132	145.5	328	339.4	11	14.3	52	47.9

SOURCE: Geoffrey Berry and Muriel L. Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," *British Journal of Industrial Medicine* 40:1 (February 1983): 4 (Table 8).

among his chrysotile miners was 104. In the Ferodo study of friction workers, the corresponding SMR was 106, and, as the authors pointed out, this slight excess could have arisen by chance; that is, it was not statistically significant.[43]

The 10 persons dying of mesothelioma in the main mortality study were examined in more detail in a case-control analysis. This showed that 8 of the 10 had definitely been exposed to crocidolite, and the authors concluded from their analysis (which sought to control for heavy chrysotile exposure) that crocidolite was predominantly responsible for the mesotheliomas.[44]

(b) Connecticut

Very recently, Dr. A.D. McDonald and her colleagues have published the results of studies in three American asbestos factories in an effort to investigate the effect of fibre type and industrial process on asbestos-related disease.[45] Two of the factories manufactured textiles and will be discussed in this chapter under that heading. The third, located in Connecticut, manufactured friction products and packings using chrysotile only until 1957, when a small amount of anthophyllite was also used. A small amount of crocidolite was also used on an experimental basis, but not until 1964.[46]

The cohort examined was defined as all males who had been employed for one calendar month or more before January 1, 1959, and who had a social security number and name that matched with data in U.S. social security files. As defined, the cohort constituted 3,641 males, of whom over 96% were traced to the end of 1977, by which time 1,267 had died. Exposure information was limited, consisting in the main until the 1970s of insurance company surveys. Before 1970, measurements were of dust, not fibres, and the authors did not attempt to convert the particle measurements to fibres due largely to their lack of confidence in the results.[47]

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[43] Ibid., pp. 6-7.

[44] Ibid., pp. 3-5. See also, Newhouse, Berry, and Skidmore, "A Mortality Study of Workers Manufacturing Friction Materials with Chrysotile Asbestos," pp. 906-908. In his testimony before this Commission, Mr. Julian Peto suggested that there was a statistical error in this case-control analysis. Mr. Peto testified that the workers who were exposed to crocidolite in the area of the plant where crocidolite was used were almost exclusively production workers who had extremely heavy exposure to chrysotile. Mr. Peto suggested that what is missing from this study is an analysis that assumes that the chrysotile exposure caused the mesothelioma and an attempt to ascertain whether one could completely remove that association by allowing for crocidolite exposure. In Mr. Peto's view, had that analysis been done, results symmetrical with those obtained by the authors, assuming that the crocidolite exposure caused the mesothelioma, would have been achieved. See RCA Transcript, Evidence of Mr. Julian Peto, 30 July 1981, Volume no. 25(B), pp. 83-86.

[45] Alison D. McDonald and John S. Fry, "Mesothelioma and Fiber Type in Three American Asbestos Factories — Preliminary Report," *Scandinavia Journal of Work, Environment and Health* 8, Supp. 1 (1982): 53-58. See also, Alison D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," *British Journal of Industrial Medicine* 40 (1983): 361-367; Alison D. McDonald et al., "Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture," *British Journal of Industrial Medicine* 40 (1983): 368-374; and Alison D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant," *British Journal of Industrial Medicine*, in press, 1984.

[46] A. D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant."

The authors did estimate that in the 1930s dust levels were generally in the range of 1 to 5 mppcf, save for certain much dustier processes (fibre preparation, fibre mixing, grinding, and finishing) where the levels were considerably higher. The average length of employment in this plant was 8.7 years, and the average dust concentration to which the men were exposed was 1.84 mppcf.[48] The exposure levels appear to have been quite similar to those at Ferodo.[49] Table 5.4 shows male deaths 20 years after first employment, by cause, in relation to duration of service.

The results of the study indicated an overall SMR of 108.5 (using Connecticut mortality rates), but the excess was not found in the longer-service employees. It occurred predominantly among those who had worked for one year or less. SMRs were increased for lung and gastrointestinal cancer, but again this was largely due to the excess in men employed less than one year. This lack of any systematic dose-response relationship in the study was also present when the data were analyzed by cumulative dust exposure, although there was some suggestion of an increasing risk for lung cancer with increasing exposure if the lowest dust category were excluded. No case of mesothelioma was found within this cohort.[50]

The authors of the study suggested that the excess mortality from all causes of death including lung cancer in the men employed less than one year may have arisen from the poorer health of those short-term employees and that otherwise the results suggest that the adverse health effects of employment in this chrysotile friction products plant were quite small.[51] While not as negative as the results at Ferodo, they are certainly compatible with those results and they are quite similar to the experience of the Quebec chrysotile miners.

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[47] Ibid., pp. 4–5.

[48] Ibid., pp. 5–6.

[49] Ibid., p. 9. The authors have stated that "The exposure levels in the two factories appear to have been fairly similar."

[50] Ibid., p. 7.

[51] Ibid., p. 8.

Table 5.4

Male Deaths 20 Years After First Employment, by Cause, in Relation to Duration of Service

Cause of Death*	Length of Gross Service (Years)									
	<1		1<5		5<20		≥20		Complete Cohort	
	O	SMR	O	SMR	O	SMR	O	SMR	O	SMR
All causes	246	129.9	189	104.0	130	104.8	238	97.2	803	108.5
Malignant neoplasms	60	144.9	50	125.7	29	114.0	63	118.3	202	126.5
Respiratory	24	180.0	19	149.4	9	122.6	21	133.4	73	148.7
Digestive	17	132.9	16	128.3	5	60.6	21	116.9	59	114.4
Other	19	120.4	25	164.9	15	190.4	21	107.2	70	115.9
Heart disease	99	125.3	79	104.9	44	83.7	100	93.1	322	102.5
Respiratory tuberculosis	0	–	0	–	0	–	4	283.3	4	145.9
Other respiratory	13	196.3	8	126.2	4	85.2	8	92.2	33	–
Pneumoconiosis	(6)	–	(3)	–	(1)	–	(2)	–	(12)	–
Cerebrovascular	18	137.6	14	108.4	15	142.7	20	102.4	67	119.6
Accidents	11	121.1	5	69.2	5	101.7	7	68.6	28	89.1
Other known	37	123.5	24	90.4	29	147.1	35	87.9	125	107.7

Note: *ICD [International Classification of Diseases] Codes, except that ICD Codes 160-164 are here grouped under "respiratory" malignant neoplasms and the "other respiratory" category includes only bronchitis, pneumonia, and pneumoconiosis (ICD 490-502, 523-524).

SOURCE: Alison D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant," *British Journal of Industrial Medicine,* in press, 1984, Table 4.

B.3 Asbestos Manufacturing — Textiles

(a) Rochdale, England

No group of asbestos workers has been more studied than those employed at the Rochdale textile factory in England. Rochdale was the subject of the first systematic epidemiological study of asbestos-related disease published by Sir Richard Doll in 1955.[52] The health experience of the workers at Rochdale formed the basis of the 1968 report of the British Occupational Hygiene Society which led to the adoption of the 2 f/cc standard in the United Kingdom.[53] The morbidity experience of these workers has continued to be studied by Mr. Geoffrey Berry and his colleagues,[54] while their mortality experience has been recently reviewed by Mr. Julian Peto.[55] We have commented elsewhere in this Report on Mr. Berry's asbestosis investigation at Rochdale.[56] Here we focus on Mr. Peto's mortality investigation.

We observe that both the type of asbestos used at Rochdale and the exposure information have been the subject of debate. There is no doubt that chrysotile asbestos has been the predominant fibre type at the plant, but there is uncertainty over the extent to which crocidolite was also used. It would, however, appear that at least some crocidolite was utilized at

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[52] Richard Doll, "Mortality from Lung Cancer in Asbestos Workers," *British Journal of Industrial Medicine* 12 (1955): 81–86. A total of 113 workers exposed to asbestos for at least 20 years were followed up; 39 deaths occurred in the group versus 15.4 expected. The excess was due to excess deaths from lung cancer (11 versus 0.8 expected) and from other respiratory and cardiovascular diseases (20 versus 7.6 expected). Ibid., Table 4, p. 84.

[53] British Occupational Hygiene Society, Committee on Hygiene Standards, "Hygiene Standards for Chrysotile Asbestos Dust," *Annals of Occupational Hygiene* 11 (1968): 54–58 [Appendix (i)]. See also, Chapter 3, Section B of this Report.

[54] Geoffrey Berry and Hilton C. Lewinsohn, "Dose-Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards, Part I. Morbidity," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 185–194; and Geoffrey Berry et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory," *British Journal of Industrial Medicine* 36 (May 1979): 98–112.

[55] Julian Peto et al., "A Mortality Study Among Workers in an English Asbestos Factory," *British Journal of Industrial Medicine* 34 (1977): 169–173; Julian Peto, "The Hygiene Standard for Chrysotile Asbestos," *The Lancet* 1 (4 March 1978): 484–489; Julian Peto, "Dose-Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards, Part II. Mortality," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 195–201; Julian Peto, "The Incidence of Pleural Mesothelioma in Chrysotile Asbestos Textile Workers," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 703–711; and Julian Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 829–836.

[56] See Chapter 2, Section D.1 of this Report.

Rochdale even as late as 1969, although the amount is not accurately known.[57]

In studies of Rochdale published prior to 1979, exposures were calculated on the basis of area samples. Area thermal precipitator measurements in millions of particles per cubic foot were taken between 1951 and 1960 and were converted to fibres per cubic centimetre using the observed ratio of parallel measurements obtained by the two methods in 1960 and 1961. From 1961 onwards, fibre levels were estimated in each area from actual membrane filter measurements. There were no routine measurements in the period 1933–1951, and exposures during this period were estimated to be as much as 50% higher than those in the early 1950s. On the basis of these data, it was estimated that the mean dust level at Rochdale was 10.8 f/cc in 1951, falling to 2.9 f/cc by 1972.[58]

Then in the late 1970s, the hygiene officers at Rochdale undertook a complete review of the exposure estimates at the plant.[59] Taking into consideration the improvements in fibre counting techniques since 1961,[60] the new survey produced substantially higher estimates of exposure levels than previously reported and indicated that average dust levels were in the region of 30 f/cc in 1951 and remained high until 1970.[61]

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[57] Mr. Peto has indicated that the asbestos exposure at Rochdale was predominantly to chrysotile, although a small proportion of crocidolite fibre was used at various times after 1933. See Peto, "The Hygiene Standard for Chrysotile Asbestos," p. 487. In a more recent publication, Mr. Peto observed that the extent of exposure to crocidolite at Rochdale is not accurately known, but appears to have been much lower than that at the East London asbestos factory studied by Newhouse and Berry. See Peto, "The Incidence of Pleural Mesothelioma in Chrysotile Asbestos Textile Workers," p. 708. In their report to the U.K. Advisory Committee on Asbestos, Dr. E. Donald Acheson and Dr. Martin J. Gardner stated that "There appears to be uncertainty and possibly a conflict of evidence about the extent and period of usage of crocidolite at Rochdale which the present authors have been unable to resolve. . . . A recent communication from Mr. H.D.S. Hardie of Turner and Newall states that between 1931 and 1970 not less than 2,500 tonnes [metric tons] of crocidolite were used at Rochdale, i.e. an average of about 60 tonnes per annum." See Acheson and Gardner, "The Ill Effects of Asbestos on Health," paragraph 119, p. 26. However, in their 1983 update, the authors now suggest that about 10,000 tonnes of crocidolite fibre in yarn were used at Rochdale between 1931 and 1970. See Acheson and Gardner, *Asbestos: The Control Limit for Asbestos*, paragraph 30, p. 5.

[58] Peto et al., "A Mortality Study Among Workers in an English Asbestos Factory," pp. 171–172; and Peto, "The Hygiene Standard for Chrysotile Asbestos," p. 487.

[59] Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," pp. 831–832.

[60] Mr. Peto has observed that the most notable improvement in counting technique was the change to graticule counting, thereby increasing the estimated fibre count by a factor of 2 or more. See Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," p. 831. Dr. Acheson testified that in his view the U.K. Advisory Committee on Asbestos was prepared to accept the fact that there had been approximately a fivefold increase in the rigorousness of the standard in the United Kingdom due to modern counting techniques (graticule vs. full view) and due to the difference between personal and area sampling. RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 55.

While the estimates of exposure have changed over the years at Rochdale and while average rather than individual estimates have been made, still this is the only asbestos manufacturing plant studied which has fibre measurements sufficiently far back in time to provide direct quantitative data that can be used for epidemiological study and risk assessment.[62]

The most recent mortality studies of Rochdale were published by Mr. Julian Peto in 1980. His total cohort of 1,106 workers was broken down into five sub-cohorts based on whether a worker was first exposed prior to 1933 or after 1933 and whether he worked more than 10 or more than 20 years at the plant. The workers first exposed after 1933 were then further subdivided between those first starting employment between 1933 and 1950 and those first starting in 1951 or later. The 679 men who entered the factory in 1933 or later and had by the end of 1972 been exposed for at least 10 years were followed to the end of 1978.[63] Table 5.5 shows the mortality experience of these 679 male asbestos textile workers.

The SMR for lung cancer, 20 years or more from first exposure, for all persons who worked at Rochdale for at least 10 years starting in 1951 or later, was approximately 500 (8 lung cancer deaths versus 1.62 expected).

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[61] Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," p. 832. The revised estimates of mean dust levels at Rochdale indicate figures of 32.4 f/cc in 1951; 23.9 f/cc in 1956; 12.2 f/cc in 1961; 12.7 f/cc in 1966; and 4.7 f/cc in 1971. The corresponding previous estimates were 10.8 f/cc in 1951; 5.3 f/cc in 1956; 5.2 f/cc in 1961; 5.4 f/cc in 1966; and 3.4 f/cc in 1971.

[62] Mr. Peto indicated in his evidence that there have been two revisions of the dust estimates at Rochdale since the publication of the 1968 British Occupational Hygiene Society report. There was the revision which led to the analysis by Berry et al. in "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory"; and the further revision, which was given to Mr. Peto and upon which he based his 1980 publication called "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory." See RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 90–91. See also, British Occupational Hygiene Society, Committee on Hygiene Standards, "Hygiene Standards for Chrysotile Asbestos Dust," Appendix (i), pp. 54–58. Mr. Peto maintained in his testimony that in his view there was no other cohort study which had as reasonably reliable evidence in terms of fibres and as moderately good measurements as the Rochdale cohort. RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 83. Dr. Finkelstein testified that in his view Mr. Peto's study, which dealt solely with lung cancer and had one data point, was the only one which provided any data for a quantitative risk assessment. See RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 112–113.

[63] Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," pp. 829–830. The breakdown of the entire cohort of 1,106 workers was described in an earlier article by Peto et al., "A Mortality Study Among Workers in an English Asbestos Factory," p. 170.

Table 5.5

Mortality Experience of 679 Male Asbestos Textile Workers

Year First Exposed	Period Since First Exposure (Years)	Man-Years	Lung Cancer		Pleural Mesothelioma		Other Cancers		Asbestosis		Other Respiratory Disease		Other Causes	
			O	E	O	Rate x 10^3	O	E	O	Rate x 10^3	O	E	O	E
1933- 1950 n = 424	10-	1,633	2	1.80	0	0.0	1	2.73	0	0.0	1	2.38	8	11.02
	15-	1,860	4	2.98	0	0.0	2	4.06	2	1.1	3	3.73	17	16.71
	20-	1,760	3	3.97	1	0.6	6	5.16	2	1.1	4	5.02	29	21.64
	25-	1,496	10	4.54	2	1.3	6	5.98	2	1.3	9	6.08	24	25.20
	30-	837	8	3.14	2	2.4	5	4.21	2	2.4	4	4.47	16	17.80
	35 +	507	1	2.20	2	3.9	4	2.93	2	3.9	5	3.06	12	12.33
	Total	8,093	28	18.63	7	–	24	25.07	10	–	26	24.73	106	104.70
1951 or later n = 255	10-	1,123	1	1.30	0	0.0	0	1.62	0	0.0	1	1.17	5	6.49
	15-	1,022	3	1.74	0	0.0	3	2.16	0	0.0	2	1.63	8	8.86
	20-	556	7	1.31	0	0.0	2	1.64	0	0.0	0	1.30	4	6.61
	25 +	96	1	0.31	0	0.0	0	0.37	0	0.0	0	0.32	1	1.52
	Total	2,797	12	4.65	0	–	5	5.80	0	–	3	4.42	18	23.28

SOURCE: Julian Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," in *Biological Effects of Mineral Fibres,* vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), Table 1, p. 830.

The average cumulative exposure for this group of employees was 100 f/cc-yrs in terms of the earlier exposure estimates and in the order of 300 f/cc-yrs in terms of the revised estimates.[64] Using these revised estimates, the SMR for lung cancer at 100 f/cc-yrs was approximately 165 (compared to 104 for the Quebec miners and 106 for the Ferodo friction workers). While admittedly the numbers are small, the mortality risk for Rochdale textile workers appears to be considerably higher than for chrysotile miners or friction product workers.

Mr. Peto has reported 14 cases of pleural mesothelioma at Rochdale among those first employed prior to 1951;[65] 7 of these cases occurred among those first employed prior to 1933. Among those first employed after 1933 but before 1951, the observed incidence of mesothelioma rose steadily from 60 per 100,000 per annum at 20 to 25 years after first employment to 400 per 100,000 per annum beyond 35 years.[66] Due to the uncertainty over the use of crocidolite at Rochdale, it was not possible to determine the type of fibres to which the mesothelioma cases were exposed. Mr. Peto has, however, commented that in his view the occasional exposure to crocidolite was unlikely to be the major cause of the incidence of mesothelioma at Rochdale.[67]

(b) Charleston, South Carolina

In September 1980, Dr. John M. Dement, an industrial hygienist at the time with NIOSH in the United States, presented findings of a study of asbestos textile workers at Charleston, South Carolina, which greatly surprised everyone familiar with the literature on asbestos.[68] The surprise was generated by two considerations: the excess disease incidence at Charleston was orders of magnitude higher than had been found in any other reported study, and yet the exposure of the workers had been to chrysotile asbestos.

The cohort reported on by Dr. Dement was limited to the 768 white males employed 6 or more months in the textile production operations of the Charleston plant with at least one month of employment between

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[64] RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 103.

[65] Peto, "The Incidence of Pleural Mesothelioma in Chrysotile Asbestos Textile Workers," p. 703.

[66] Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," p. 831.

[67] Peto, "The Incidence of Pleural Mesothelioma in Chrysotile Asbestos Textile Workers," p. 708. See, however, note 57, *supra*.

[68] John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," *Annals of Occupational Hygiene* 26:1–4 (1982): 869–887. This paper was first presented at the Fifth International Symposium on Inhaled Particles at Cardiff, Wales, in September 1980. See also, John M. Dement et al., "Exposures and Mortality Among Chrysotile Asbestos Workers. Part I: Exposure Estimates," *American Journal of Industrial Medicine* 4 (1983): 399–419.

January 1, 1940 and December 31, 1965. The cohort was followed through 1975, thus ensuring a minimum latency period of 10 years.[69]

There were 5,952 measurements collected in the period 1930–1975, although all but 376 were collected after 1959. Prior to 1965, all measurements were in dust particles by impinger; from 1965 to 1971, fibre measurements by membrane filter were also made; and, in 1971, the impinger method was entirely replaced by the membrane filter method. Dr. Dement converted his dust particle counts to fibre counts on the basis of two sources of data: 120 paired impinger membrane filter samples collected in the plant by the U.S. Public Health Service in 1965 and 986 paired samples collected in the plant in the period 1968–1971. On the basis of these data Dr. Dement used a conversion factor of 3 f/cc to 1 mppcf for all textile operations save preparation, for which a conversion factor of 8 was used.[70]

It is clear that chrysotile was the only asbestos received at Charleston as a raw fibre. Crocidolite was used in weaving from the 1950s until approximately 1975, but the amount was trivial, the weaving was done by a wet process, and crocidolite was never carded, spun, or twisted.[71]

Table 5.6 sets out the observed and expected deaths, by cause, for the white male asbestos textile workers in the cohort. In the entire cohort there were 191 observed deaths versus 141.84 expected, for an SMR of 135; there were 26 lung cancer deaths versus 7.47 expected, for an SMR of 348; and there were 15 deaths from asbestosis. Smoking data tended to show that the prevalence of smoking among men in the cohort was nearly identical to that of U.S. white males as a whole.[72]

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[69] Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," p. 870.

[70] Ibid., pp. 871–872; and Dement et al., "Exposures and Mortality Among Chrysotile Asbestos Workers. Part I: Exposure Estimates," pp. 408–409.

[71] Dement et al., "Exposures and Mortality Among Chrysotile Asbestos Workers. Part I: Exposure Estimates," p. 400. The authors have indicated that the total amount of crocidolite ever processed at the Charleston plant was less than 2,000 pounds. They also stated that according to company personnel, an average of approximately 6 to 8 million pounds of chrysotile was processed annually. See also, RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, pp. 91–100. Dr. Dement also observed in his evidence that exposure to crocidolite could not have had a significant effect on lung cancer mortality in that almost all of the lung cancer deaths occurred 30 or more years from first exposure.

[72] Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," pp. 880–881. Among the asbestos workers for whom smoking data were available, 52.4% were found to be current smokers, 25.3% were non-smokers, and 22.3% were past smokers. The corresponding figures for U.S. white males in 1965 were 51.5%, 26.4%, and 22.1%.

Table 5.6

Observed and Expected Deaths, by Cause, for White Male Asbestos Textile Workers, 1940–1975

Cause of Death	ICDA 7th List Number	Obs	Exp	SMR
All causes		191	141.84	135†
Malignant neoplasms		43	24.66	174†
Digestive system	150–159	9	7.10	127
Trachea, bronchus, and lung	162–163	26	7.47	348†
Other and unspecified sites		8	10.09	79
Disease of the central nervous system	330–334, 345	9	7.72	117
Disease of the circulatory system	400–468	58	57.43	101
All tuberculosis	001–019	4	2.77	144
Non-malignant respiratory disease		18	6.85	263†
Acute upper respiratory infection	470–475	0	0.04	–
Influenza	480–483	0	0.37	–
Pneumonia	490–493	0	2.98	–*
Bronchitis	500–502	0	0.41	–
Other respiratory diseases	510–527	18	3.05	590†
Accidents	800–962	14	16.64	84
Other violent deaths	963–964 970–985	7	6.10	115
All other known causes		23	17.92	128
Unknown causes		15	1.75	–

Notes: *Significant at $p < 0.05$.
†Significant at $p < 0.01$.

SOURCE: John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," *Annals of Occupational Hygiene* 26:1–4 (1982): 873 (Table 3).

Table 5.7 is a summary of dose-response relationships for selected causes of death. Dose-response analysis was limited to those workers who had achieved 15 or more years since first employment. Cumulative exposure was divided into four exposure categories, and for lung cancer the SMR rose from 223 in the lowest category to 1,553 in the highest, with an overall SMR of 399. Corresponding overall SMRs for deaths from all causes and from non-malignant respiratory disease were 151 and 642 respectively. Despite the extraordinarily elevated lung cancer risk, some 50 times greater than that found among friction product workers or chrysotile miners, there was only one mesothelioma death (peritoneal) in this cohort, and the incidence of gastrointestinal cancer was only slightly increased, with an SMR of 127.

Dr. Dement's study has been subjected to rigorous scrutiny by many of the world's acknowledged experts on asbestos in an effort to explain and qualify his findings.[73] Several concerns about his study were voiced during our hearings by other witnesses. We list these concerns and comment on them as follows:

(i) The Charleston workers may have had prior shipyard exposure.[74] While Dr. Dement did not have a prior occupational history on his entire cohort, he did have past employment histories on 10 of the 26 lung cancer cases and none of them had shipyard experience. Furthermore, the Charleston shipyard used crocidolite and Dr. Dement observed that if a high proportion of his textile workers had been employed in the shipyards, he would have expected to see more than one mesothelioma.[75]

(ii) There were few measurements prior to 1960.[76] While this is obviously true, compared to other studies the quantity of these measurements is appreciable. Moreover, textile operations have not changed a great deal over time, and according to Dr. Dement's evidence before this Commission, insurance company measurements, government measurements, and the company's own measurements were quite consistent.[77]

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[73] For example, Dr. Gibbs testified that he and a number of other scientists met with Dr. Dement to look at the Charleston data to determine whether there was any explanation as to why the risks in relation to exposure appeared to be so different from those found in all other studies. See RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 60–61.

[74] See, for example, RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 45.

[75] RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 12.

[76] For example, Mr. Peto testified that the Dement study suffered from the fact that most of the excess mortality was in workers who were first exposed in the 1930s and 1940s when no reliable measurements were taken. See RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 145.

[77] RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, pp. 14–15.

Table 5.7

Summary of Dose-Response Relationships, for Selected Causes, Among White Males Achieving 15 or More Years' Latency

Cumulative Dose (Fibre cm^{-3} Days)	All Causes			Diseases of the Circulatory System (400-458)			Lung Cancer (162, 163)			Digestive System Cancer (150-159)			Other Non-malignant Respiratory Diseases (510-527)		
	Obs	Exp	SMR	Obs	Exp	SMR	Obs	Exp	SMR	Obs	Exp	SMR	Obs	Exp	SMR
<10,000	65	56.17	118*	21	24.37	86	8	3.59	223*	1	2.73	37	1	1.41	71
10,000-40,000	57	32.83	174†	23	16.94	136*	7	1.96	357†	3	1.85	162	7	0.78	897†
40,000-100,000	32	13.63	235†	7	6.37	110	9	0.92	978†	3	0.79	380	7	0.38	1,842†
100,000-200,000	6	3.42	175	1	1.30	77	2	0.13	1,553*	0	0.16		2	0.08	2,500*
Total	160	105.06	151†	52	48.98	106	26	6.51	399†	7	5.53	127	17	2.65	642†

Notes: *Significant at $p<0.05$.
†Significant at $p<0.01$.

SOURCE: John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," *Annals of Occupational Hygiene* 26:1-4 (1982): 877 (Table 7).

(iii) The estimated exposures were far too low in light of the pictorial evidence of prior conditions in the plant.[78] This factor is difficult to assess, but we note that Dr. A.D. McDonald, who has studied the same plant using the same exposure information, indicated that the estimates were reasonably consistent among several groups of observers and similar to levels in other American textile plants and probably also to Rochdale.[79]

(iv) Dr. Dement's conversion ratios from particles to fibres were too low, thereby overestimating the risk.[80] It does appear that the conversion factors used by Dr. Dement were on the low side of the range of conversions done by Ayer, Lynch, and Fanney for the U.S. Public Health Service in 1965.

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[78] In his testimony, Dr. Hans Weill indicated that Dr. Dement himself had shown a slide of a carding machine in the Charleston plant in the 1930s, and the slide indicated that conditions were very very dusty. Yet, according to Dr. Weill, in the published paper Dr. Dement assessed the average exposure at that time in the particular job as 5 f/cc, which Dr. Weill found a little hard to believe. See RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 105–106. Dr. A.D. McDonald, who has studied the same plant, observed in her testimony that the quantitative data on past exposure is quite perplexing in that they are relatively low compared to what one might have expected from some of the pictorial evidence. See RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 25, 62. In their paper, Dr. A.D. McDonald and her colleagues discussed the possibility that both Dr. Dement and her team had failed to give sufficient weight to the occasional extremely high exposure which, from all accounts, many employees experienced during overtime work. In the view of Dr. A.D. McDonald and her colleagues, these casual exposures, although significant, were unlikely to explain a more than twofold error in cumulative exposure estimates. See A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," p. 366.

[79] A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," p. 366.

[80] For example, Dr. Weill suggested in his evidence that a conversion factor of 3 was a little too low because the simultaneous samplings of the U.S. Public Health Service suggested at least a 6 to 1 ratio if not higher for textile manufacturing. Dr. Weill observed that in textile manufacturing asbestos is a very much higher proportion of the total dust than, for example, in asbestos-cement manufacturing. See RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 101–102. Dr. Gibbs also felt that Dr. Dement's conversion ratios might have been a little lower than he would have expected. See RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 60–61. In their own review of the environmental measurements, Dr. A.D. McDonald and her colleagues observed a conversion range of 1.3 to 10.0, with an average of about 6, which was the same figure that Ayer, Lynch, and Fanney of the U.S. Public Health Service concluded might be appropriate for textile processes. See A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," p. 366. See also, Howard E. Ayer, Jeremiah R. Lynch, and Julius H. Fanney, "A Comparison of Impinger and Membrane Filter Techniques for Evaluating Air Samples in Asbestos Plants," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 274–287.

Nonetheless, both Mr. Howard E. Ayer himself and Dr. A.D. McDonald considered the conversion factors used by Dr. Dement to be reasonable.[81]

(v) Dr. Dement has used an unorthodox methodology to calculate his exposure-response relationships.[82] As Dr. Murray M. Finkelstein observed, it would appear that the methodology used by Dr. Dement tended to underestimate the risk at low exposure categories and overestimate the risk at higher exposure categories. Still, his methods do not detract from the overall results,[83] and, as Dr. Finkelstein also observed, Dr. Dement's study appears to be a thoughtful and intelligent approach to the problem of exposure assessments.[84]

(vi) Dr. Dement used national mortality rates for his control population whereas had he used local mortality rates, his excess mortality would have been less since Charleston and the surrounding counties have a lung cancer mortality rate considerably above the national average.[85] Dr. Dement discussed his choice of national rates in his published report. He rejected the use of local rates because many citizens working in the local shipyard industry would have been exposed to asbestos and because the Charleston plant itself would have contributed to the high local cancer rate. However, even if lung cancer mortality rates for the contiguous counties were used, expected mortality rates would increase by only 15%, not enough to account for the observed excess in the cohort.[86]

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[81] See RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, p. 21; and RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 60-61. Dr. J.C. McDonald testified that Dr. Dement is an occupational hygiene engineer and is probably one of the most competent people attempting to do conversions from particles to fibres. See RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 59. Dr. Dement, in his own testimony, admitted that his conversions were on the low side of the range of conversions done by Ayer, Lynch [and Fanney] for the U.S. Public Health Service. However, Dr. Dement's conversions were certainly within the range of those conversions. See RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 33.

[82] Dr. Finkelstein testified that Dr. Dement and his colleagues had adopted a method whereby workers move from one exposure category to the next as exposure accumulates. However, person-years of risk are left behind in former categories and begin to be freshly contributed to the new category as it is entered. Dr. Finkelstein suggested that this is not an appropriate method when one is dealing with mortality from diseases of long latency. According to Dr. Finkelstein, as a result of using this methodology, too many deaths and too few person-years of risk would be attributed to the higher exposure categories and too few deaths to the lower exposure categories. The net result is an overestimate of the slope of the dose-response curve. See RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 113-116. See also, letter from Dr. Murray M. Finkelstein to the Editor, *American Journal of Industrial Medicine*, 27 May 1983. This letter, which contains Dr. Finkelstein's critique of the methodology used by Dr. Dement and his colleagues, is to be published in the *American Journal of Industrial Medicine*.

[83] Dr. Dement, in his testimony, indicated that his methodology did tend to underestimate the risk at lower levels and overestimate it at higher levels, but only slightly. The alternative method, for example, used by Dr. Finkelstein in estimating dose, would be to have a person accumulate exposure up to a fixed point, say, 20 years, cut off cumulative exposure at that point, and then have a follow-up period. According to Dr. Dement, each of these two methods has its own advantages. See RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, pp. 29-31.

[84] RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 55.

(vii) Dr. Dement has not included black males in his cohort. He has not done so apparently because of the poor trace rate and cause of death information for this group. Still, as Dr. Dement has pointed out, at least in terms of duration of employment, the black male employees have elevated SMRs fairly consistent with the white male data.[87]

We have devoted considerable space to a discussion of the Charleston study. We have done so because of the serious implications raised by its startling results. In the end, we are driven to the general conclusion that some or all of the various comments or criticisms which have been made of the study may account for part of the difference between it and other studies, but even together they cannot account for all of the difference.[88]

Cogent confirmation of Dr. Dement's findings may be found in a subsequent and independent investigation of the health experience at the Charleston plant by Dr. A.D. McDonald and her associates.[89] Their cohort was considerably larger than Dr. Dement's, comprising in all 2,543 men employed for at least one month between 1938 and 1958 and followed to the end of 1977, by which time 863 employees or 34% of the cohort had died. Of the 863 deaths, there were 6 cases for which the age at death was not known. The authors' man-years calculations were, therefore, based upon 857 deaths, and this figure is used in Table 5.18 in this chapter. In calculating SMRs, only deaths 20 or more years from first employment were included. Table 5.8 shows male deaths 20 years after first employment, by cause, in relation to duration of service. For the entire cohort as defined, the SMR for all causes of death was 127.4; for lung cancer, 199.5; and for abdominal cancer, 151.7 (the latter figure being higher than Dr. Dement's corresponding figure for gastrointestinal cancer). There were 20 deaths from pneumoconiosis, all among men with 20 or more years' employment, and there was only one mesothelioma death in the entire cohort, the same case reported by Dr. Dement.[90] When calculated according to cumulative dust exposure, the SMR for lung cancer rose sharply, reaching 1,031.9 (on the basis of 8 cases) in the highest dust category, and the relationship was essentially linear. Table 5.9 sets out male deaths 20 years after first employment, by cause, in relation to dust exposure accumulated up to 10 years before death.

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[85] RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 36–37. Lung cancer death rates for South Carolina were nearly equal to U.S. rates. On the other hand, rates for the county in which the Charleston plant was located were 75% higher than U.S. rates for white males.

[86] Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," pp. 879–880.

[87] See RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 81. Dr. Dement also observed in his evidence that the entire population of workers at the Charleston plant was approximately 10,000, but that included both textile and non-textile workers, black and white, male and female workers. See ibid., p. 40.

[88] Dr. A.D. McDonald reached similar conclusions. RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, p. 63. In their paper, Dr. A.D. McDonald and her colleagues suggested that even giving effect to all of the critiques, it was improbable that the fiftyfold difference in exposure-response between the Quebec miners cohort and the Charleston cohort could be reduced to less than about tenfold. See A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," p. 366.

[89] A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant."

Considering that Dr. Dement excluded from his cohort clerical employees and employees who had worked less than 6 months, whereas Dr. A.D. McDonald did not, the findings of the two studies are almost identical.[91] In seeking to compare them with the quite different results in chrysotile mining and friction products manufacturing, two further observations may be made. First, the average dust concentrations at Charleston, although low when compared with mining and milling, were in the same range as the Connecticut friction products plant in which little or no excess lung cancer mortality occurred — 1.80 mppcf compared with 1.84 mppcf.[92] Second, there were no major differences in smoking habits as between the textile workers and the Quebec miners and millers that might explain the different incidence of lung cancer in the two cohorts.[93]

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[90] Ibid., p. 365. The one death was a man born in 1904 who died in 1967. He was first employed at the plant in 1925, worked as a mule spinner from 1933 to 1955 and as an oven helper until he left in 1965. There was no autopsy. In her testimony, Dr. A.D. McDonald indicated that she had considered whether there were cases of mesothelioma occurring at Charleston that had not been detected. She observed that the Department of Pathology at the Medical College of South Carolina at Charleston is very interested in mesothelioma and has been looking very hard for cases and has yet not found any save for the one case reported by her and by Dr. Dement and one other case that did not appear in either cohort. See RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 48–49.

[91] A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," p. 366 and Figure 2, p. 366.

[92] Ibid., Table 3, p. 363; and A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant," Table 3.

[93] A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," pp. 366–367. Dr. A.D. McDonald and her colleagues noted that the dose-response line at Charleston is 50 times steeper than the dose-response line for the Quebec mining cohort.

Table 5.8

Male Deaths 20 Years After First Employment, by Cause, in Relation to Duration of Service

Cause of Death*	Length of Gross Service (Years)									
	<1		1<5		5<20		≥20		Complete Cohort	
	O	SMR	O	SMR	O	SMR	O	SMR	O	SMR
All causes	159	107.4	113	122.7	120	156.1	178	136.7	570	127.4
Malignant neoplasms										
Respiratory	8	78.2	10	163.9	15	304.1	26	317.3	59	199.5
Abdominal	6	107.9	5	146.4	7	240.3	8	151.4	26	151.7
Other	12	130.2	7	124.9	9	196.9	7	46.2	35	127.5
Heart disease	69	108.9	34	87.6	45	141.7	70	120.8	218	113.7
Respiratory tuberculosis	1	231.8	1	347.8	1	307.9	1	131.5	4	222.8
Other respiratory	3	53.3	3	85.6	2	78.3	27	557.5	35	207.3
Pneumoconiosis	(0)	—	(0)	—	(0)	—	(20)	—	(20)	—
Cerebrovascular	9	83.0	14	193.0	6	107.3	9	76.2	38	107.2
Accidents	18	121.2	8	89.7	5	75.8	9	85.0	40	97.0
Other known	30	116.9	28	175.5	23	177.7	21	92.3	102	132.4
Not known	3		3		7		0		13	

Note: *ICD Codes, except that ICD Codes 160-164 are here grouped under "respiratory" malignant neoplasms and the "other respiratory" category includes only bronchitis, pneumonia, and pneumoconiosis (ICD 490-502, 523-524).

SOURCE: Alison D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," *British Journal of Industrial Medicine* 40 (1983): 364 (Table 4).

Table 5.9

Male Deaths 20 Years After First Employment, by Cause, in Relation to Dust Exposure (Million Particles Per Cubic Foot-Years) Accumulated to 10 Years Before Death

Cause of Death*	Dust Exposure (mppcf-yrs)									
	<10		10<20		20<40		40<80		>80	
	O	SMR	O	SMR	O	SMR	O	SMR	O	SMR
All causes	376	115.5	55	125.5	63	156.9	43	170.8	33	264.4
Malignant neoplasms										
Respiratory	31	143.1	5	182.7	8	304.2	7	419.5	8	1,031.9
Abdominal	14	114.9	4	231.6	4	247.0	4	383.6	0	—
Other	28	140.0	3	109.2	1	44.9	0	—	3	383.5
Heart disease	143	103.5	28	143.6	29	166.6	10	88.6	8	149.9
Respiratory tuberculosis	3	264.4	0	—	0	—	1	634.4	0	—
Other respiratory	8	65.9	2	119.5	6	421.7	13	1,407.8	6	1,296.0
Pneumoconiosis	(0)	—	(0)	—	(3)	—	(9)	—	(8)	—
Cerebrovascular	29	115.3	2	50.0	4	124.4	2	93.4	1	99.8
Accidents	31	99.2	2	54.1	5	152.9	1	49.4	1	120.0
Other known	79	140.4	9	116.9	4	630	5	111.5	5	236.3
Not known	10		0		2		0		1	

Note: *ICD Codes, except that ICD Codes 160-164 are here grouped under "respiratory" malignant neoplasms and the "other respiratory" category includes only bronchitis, pneumonia, and pneumoconiosis (ICD 490-502, 523-524).

SOURCE: Alison D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," *British Journal of Industrial Medicine* 40 (1983): 364 (Table 5).

(c) Lancaster, Pennsylvania

Dr. A.D. McDonald and her colleagues have also recently reported on another cohort of asbestos workers employed in a factory in rural Pennsylvania which manufactures mainly textile products but also some friction materials.[94] According to the authors, chrysotile was the principal type of asbestos used in the factory but crocidolite and amosite were also used from 1924 onwards, with the latter being utilized in particularly large quantities during World War II to make insulation blankets for the U.S. Navy.

This cohort was constituted in exactly the same way as the cohorts in the Charleston and Connecticut factory studies and in the Pennsylvania plant comprised 4,137 men, of whom 1,400 had died by the end of 1974. Dust levels at the plant appear to have been somewhat higher than in the Charleston or Connecticut plants, with average concentrations of 2.32 mppcf.[95] Table 5.10 shows male deaths 20 years after first employment, by cause, in relation to duration of service. The SMR for all causes of death, 20 years after first employment, was 109. The SMR for lung cancer among long-term employees was 158.9 and among those in the highest dust category was 416.1.[96] A comparison of the relative risks of death from all causes, from lung cancer, and from pneumoconiosis in the Charleston plant and in this Pennsylvania plant produced reasonably similar results.[97] What was not similar, however, was the mesothelioma incidence: in this plant, there were 14 deaths from mesothelioma according to death certificate information (out of 1,392 deaths, equivalent to 1%) compared to only one case in Charleston (out of 857 deaths, equivalent to 0.1%).[98] Moreover, the authors have raised the possibility that additional cases of peritoneal mesothelioma may have gone unrecognized and undiagnosed in the cohort.[99] We observe in any event that the authors have attributed the incidence of mesothelioma in this cohort to amphibole exposure, and predominantly to

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[94] A.D. McDonald et al., "Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture."

[95] Ibid., pp. 368–370 and Table 3, p. 370.

[96] Ibid., Table 5, p. 371.

[97] The SMRs in the Pennsylvania plant were somewhat depressed relative to the SMRs at Charleston. The authors have tentatively suggested that a possible reason was that the employees had smoked fewer cigarettes than the general population of the state, perhaps because of the high proportion of Mennonites in the local community. However, no firm conclusions could be drawn.

[98] A.D. McDonald et al., "Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture," pp. 372–373.

[99] Ibid., p. 372. The authors noted that of the 14 mesothelioma deaths, 10 were pleural and 4 peritoneal. All 14 deaths occurred in the period from 1960 to 1975. Another 30 deaths, 15 or more years after first employment, were given the ICD Code 199 (malignant neoplasms of other and unspecified sites). Seventeen of these 30 deaths occurred before 1965, the year after which the majority of the mesothelioma deaths occurred. The diagnosis given in many of the cases did not preclude the possibility of an unrecognized peritoneal mesothelioma.

Table 5.10

Male Deaths 20 Years After First Employment, by Cause, in Relation to Duration of Service

Cause of Death*	Length of Gross Service (Years)									
	<1		1<5		5<20		≥20		Complete Cohort	
	O	SMR	O	SMR	O	SMR	O	SMR	O	SMR
All causes	171	87.2	154	106.2	187	104.5	383	127.2	895	109.0
Malignant neoplasms										
Respiratory	9	69.6	3	32.9	14	128.8	27	158.9	53	105.0
Abdominal	8	72.9	11	133.7	11	105.9	24	131.3	54	112.7
Other	19	132.4	16	152.0	15	118.5	32	155.3	82	141.1
Heart disease	77	92.7	77	125.1	78	100.2	153	115.7	385	108.5
Respiratory tuberculosis	0	—	1	133.4	0	—	2	67.3	3	51.7
Other respiratory	4	54.2	2	38.1	11	161.0	50	442.4	67	215.0
Pneumoconiosis	(2)	—	(1)	—	(10)	—	(46)	—	(59)	—
Cerebrovascular	7	54.9	10	106.5	10	77.7	20	87.6	47	81.2
Accidents	13	117.5	15	181.1	8	87.2	9	60.1	45	103.5
Other known	30	75.2	15	52.4	37	103.0	62	103.1	144	87.2
Not known	4		4		3		4		15	

Note: *ICD Codes, except that ICD Codes 160-164 are here grouped under "respiratory" malignant neoplasms and the "other respiratory" category includes only bronchitis, pneumonia, and pneumoconiosis (ICD 490-502, 523-524).

SOURCE: Alison D. McDonald et al., "Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture," *British Journal of Industrial Medicine* 40 (1983): 371 (Table 4).

amosite, although there was no specific exposure information reported on the individual cases.[100]

The mortality experience at this Pennsylvania plant has also been investigated by NIOSH.[101] Although the cohort was somewhat smaller (comprising 3,276 workers, excluding clerical employees, employed one year or more between 1940 and 1967), the mortality results for asbestos-related disease were consistent with those found by Dr. A.D. McDonald and her colleagues. Table 5.11 shows the observed and expected deaths, according to cause, among the 2,722 white male employees in the NIOSH study cohort. The overall SMR for lung cancer among the male employees was 136. There were 17 mesotheliomas recorded in the entire cohort, 13 among male employees and 4 among female employees.[102] A noteworthy finding of the study is the high rate of suicide. The major difference between the NIOSH study and that of Dr. A.D. McDonald and her colleagues relates to the amount of amphibole asbestos each reported as being used at the plant. As we have indicated, Dr. A.D. McDonald, on the basis of her research, concluded that crocidolite and amosite were used quite extensively.[103] NIOSH, on the other hand, claimed that in all but three years during World War II, chrysotile constituted over 99% of the asbestos processed; and while in those three years amosite was used for naval specifications, it still only accounted for 5% of asbestos consumption at the plant.[104] We are in no position to resolve this difference, but we do observe that in none of the 17 mesothelioma cases reported in the NIOSH study have the authors been able to isolate exposure to chrysotile alone.

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[100] Ibid., p. 373.

[101] See Joseph K. Wagoner, William M. Johnson, and Richard A. Lemen, "Malignant and Nonmalignant Respiratory Disease Mortality Patterns Among Asbestos Production Workers," in U.S. Congress, Senate, *Congressional Record*, 93rd Cong., 1st sess., 14 March 1973, vol. 119, pt. 6, pp. 7828–7830. This study was updated by Cynthia F. Robinson, Richard A. Lemen, and Joseph K. Wagoner, "Mortality Patterns, 1940–1975 Among Workers Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility," in *Dusts and Disease*, eds. Richard A. Lemen and John M. Dement (Park Forest South, Illinois: Pathatox Publishers, Inc., 1979), pp. 131–143.

[102] Robinson, Lemen, and Wagoner, "Mortality Patterns, 1940–1975 Among Workers Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility," pp. 138–139.

[103] Dr. A.D. McDonald and her colleagues indicated that crocidolite and amosite were used at the plant from 1924 onwards. They noted that much of the crocidolite was imported as yarn and only about 3 to 5 tons of raw fibre were used annually, but that amosite was extensively used, reaching a peak of 600 tons in 1943. According to the authors, chrysotile usage varied between 3,000 and 6,000 tons annually. See A.D. McDonald, "Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture," p. 369.

[104] Robinson, Lemen, and Wagoner, "Mortality Patterns, 1940–1975 Among Workers Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility," p. 133.

Table 5.11

The Observed and Expected Deaths, According to Cause, Among White Males Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility

Cause of Death	List Number*	Observed	Expected	SMR
Malignant neoplasms	140-205	168	128.7	131***
Digestive system	150-159	50	41.4	121
Bronchogenic cancer	162-163	49	36.1	136**
Other and unspecified	140-149, 156B, 161, 164, 165, 170-199, 200-205	69	51.2	135**
Vascular lesions affecting the central nervous system	330-334, 345	49	57.0	86
Diseases of the heart	400-443	375	315.4	119**
Non-malignant respiratory disease	470-527	92	38.0	242***
Influenza, pneumonia, bronchitis, and acute upper respiratory infection	470-475, 480-483, 490-493, 500-502	16	21.4	74
Other respiratory disease	510-527	76	16.4	463***
Suicides	963, 970-979	30	17.1	175***
Other violent deaths	800-962, 964, 980-985	45	59.3	76
All other known causes		138	125.8	110
Unknown causes		15	0	—
Total		912	741.3	123**

Notes: *Seventh Revision of the International Lists of Diseases and Causes of Death [now International Classification of Diseases].
**Significant at $p<0.05$.
***Significant at $p<0.01$.

SOURCE: Cynthia F. Robinson, Richard A. Lemen, and Joseph K. Wagoner, "Mortality Patterns, 1940-1975 Among Workers Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility," in *Dusts and Disease,* eds. Richard A. Lemen and John M. Dement (Park Forest South, Illinois: Pathatox Publishers, Inc., 1979), Table 2, p. 135.

B.4 Asbestos Manufacturing — Cement Products

(a) New Orleans, Louisiana

Dr. Hans Weill and his colleagues at Tulane University have now reported on the mortality experience of workers in two asbestos-cement building materials plants in New Orleans.[105] Chrysotile was the principal fibre used in both plants, but crocidolite was also used in the pipe department of the second plant where it constituted 3% of the final product. Dr. Weill has reported that the total percentage of asbestos fibre in most asbestos-cement products ranges from 15 to 28%,[106] so that crocidolite constituted between 10 and 20% of the asbestos used to make cement pipe.

As originally identified, the cohort studied by Dr. Weill included all workers employed in either of the two plants for at least one month prior to January 1, 1970. In order to maximize the likelihood of detecting adverse health effects, results were reported for the 5,645 men with a minimum follow-up through 1974 of 20 years from first employment.

Using social security records, Dr. Weill was only able to trace 75% of the cohort, 11% of whom had died. Dr. Weill's study has been criticized for its relatively poor trace rate, and it would appear that with the vital status of one-quarter of the cohort unknown, the possibility for bias in the results exists.[107] Moreover, as Dr. Weill assumed that all those not known to be dead were alive, it is quite possible that the disease risk has been underestimated in his study. On the other hand, Dr. Weill's trace rate was best among those most heavily exposed and where one would expect excess disease to be greater. Further, as Dr. Weill has pointed out, there is no necessity to assume any relationship between cause-specific mortality and trace rate.[108] In any event, Dr. Weill is currently updating his study and apparently has been able to trace a much larger percentage of his cohort.[109] Unfortunately, his updated results were not available in published form at the time this Report was being written.

In terms of the published study, no excess mortality occurred in any exposure group (measured in dust particles) for any cause other than lung cancer. In turn, these rates showed excess risk only at exposure levels over 100 mppcf-yrs. Table 5.12 indicates standard mortality ratios, by cause of death, within various dust exposure categories.

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[105] Hans Weill, Janet Hughes, and Carmel Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," *American Review of Respiratory Disease* 120:2 (August 1979): 345–354.

[106] Ibid., p. 346.

[107] See, for example, RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 65; and Geoffrey Berry, "Discussion Summary," in *Biological Effects of Mineral Fibres*, vol. 2, p. 862.

[108] RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 68–70.

[109] Letter from Dr. Hans Weill, Pulmonary Diseases Section, Department of Medicine, Tulane Medical Center, New Orleans, Louisiana to the Royal Commission on Asbestos, 11 April 1983.

The overall lung cancer mortality experienced by persons employed 10 or more years exhibited an SMR of 215 which, as Dr. Weill and his colleagues have pointed out, is remarkably similar to the corresponding figure of 217 found by Mr. Peto in his earlier 1978 mortality study at Rochdale.[110] There were only 2 mesotheliomas (both pleural) reported in the study, one employed only 10 months with known exposure only to chrysotile and the other employed for 14 years in the pipe plant thereby having exposure to both chrysotile and crocidolite.[111] However, Dr. Weill has recently advised us that more cases of mesothelioma have begun to appear in this cohort.[112]

In the published study, Dr. Weill separately analyzed the risk of lung cancer by fibre type. Among the 4,201 workers not exposed to crocidolite the SMR for lung cancer was only 77 (30 deaths versus 38.8 expected). Among the 235 maintenance workers intermittently exposed to crocidolite in the pipe plant the SMR was 304 (statistically significant at the 95% confidence interval), and among the 1,004 workers steadily employed in the pipe plant with crocidolite exposure the SMR was 155.[113] Leaving aside mesothelioma, the results of the Weill study appear to suggest that exposure to crocidolite and chrysotile in combination enhances the risk of lung cancer as compared to exposure to chrysotile alone, particularly for those workers exposed intermittently in maintenance jobs.

(b) Manville Cement Workers

In 1972–1973 and again in 1979, Dr. Philip E. Enterline and Ms. Vivian L. Henderson reported on the mortality experience of a group of men, aged 65 and over, who completed their working lifetimes as production or maintenance-service employees with the Johns-Manville

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[110] Weill, Hughes, and Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," p. 353.

[111] Ibid., p. 349.

[112] Letter from Dr. Hans Weill to the Royal Commission on Asbestos, 11 April 1983.

[113] Weill, Hughes, and Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," pp. 351–352.

Table 5.12

Standard Mortality Ratios, by Cause, Within Exposure Categories

	Total Dust Within 20 Years of Initial Exposure (mppcf-yrs)*									
	≤ 10 (n = 3,037)		11-50 (n = 1,303)		51-100 (n = 383)		101-200 (n = 344)		> 200 (n = 578)	
Cause of Death	O/E**	SMR***	O/E	SMR	O/E	SMR	O/E	SMR	O/E	SMR
All causes	259/433.7	60	141/218.9	64	56/75.1	75	42/52.3	80	103/110.1	94
All malignant neoplasms (140-209)	54/77.3	70	27/37.1	73	7/12.9	54	14/9.5	147	18/19.8	91
Digestive system (150-159)	10/24.6	41	10/11.9	84	3/4.2	71	0/3.0	–	2/6.4	31
Respiratory system (160-163)	19/24.7	77	8/11.4	70	1/3.8	26	9/3.1	290†	14/6.2	226†
Other (residual)	25/28.0	89	9/13.8	65	3/4.9	61	5/3.4	147	2/7.2	28
Major cardiovascular diseases (390-448)	129/215.7	60	78/113.9	68	33/40.1	82	14/26.5	53	61/57.4	106
All other causes	76/140.7	54	26/47.8	54	12/16.2	74	9/11.6	78	20/23.8	84

Notes: * Million particles per cubic foot-years.

** Ratio of observed to expected deaths.

*** Standard mortality ratio.

† Significant at $p < 0.01$ (number of observed deaths compared to the number expected, assuming a Poisson distribution).

SOURCE: Hans Weill, Janet Hughes, and Carmel Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," *American Review of Respiratory Disease* 120:2 (August 1979): 348 (Table 2).

Corporation, who retired during the years 1941–1967, and who were exposed to asbestos dust in the course of their employment.[114] In their earlier report, the authors followed a total cohort of 1,464 retirees through to the end of 1969, by which time 822 had died. Of this cohort, 316 men who worked in the manufacture of cement shingles and sheets using only chrysotile asbestos had an overall lung cancer SMR of 142.9. A total of 106 retirees employed in the manufacture of asbestos-cement pipe using a combination of chrysotile and crocidolite had a lung cancer SMR of 588.2.[115] As this earlier investigation by Enterline and Henderson included Canadian employees, the asbestos-cement pipe workers undoubtedly included those retirees from the Johns-Manville plant at Scarborough, Ontario, who met the eligibility requirements to be part of the cohort.

In their more recent report, the authors excluded Canadian employees and thus had a cohort of 1,075 retirees averaging 25 years' employment, followed through to the end of 1973, by which time 781 men had died. Of this cohort, 402 retirees were identified as having been employed in the asbestos-cement industry: 305 in the production of rigid shingles and sheets using only chrysotile asbestos and 97 in the production of asbestos-cement pipe using both crocidolite and chrysotile asbestos.[116] Exposure levels were estimated on the basis of hygiene surveys, discussions with long-service employees, and a judgement as to the effect of changes in conditions. For the entire cohort, the SMR for lung cancer was 270.4.[117] While there were many Manville employees who died of mesothelioma prior to age 65, only 5 qualified for inclusion in the cohort, having died after age 65.[118]

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[114] Philip E. Enterline, Pierre DeCoufle, and Vivian L. Henderson, "Mortality in Relation to Occupational Exposure in the Asbestos Industry," *Journal of Occupational Medicine* 14:12 (December 1972): 897–903; Philip E. Enterline, Pierre DeCoufle, and Vivian L. Henderson, "Respiratory Cancer in Relation to Occupational Exposures Among Retired Asbestos Workers," *British Journal of Industrial Medicine* 30:1 (January 1973): 162–166; Philip E. Enterline and Vivian L. Henderson, "Type of Asbestos and Respiratory Cancer in the Asbestos Industry," *Archives of Environmental Health* 27:5 (November 1973): 312–317; and Vivian L. Henderson and Philip E. Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 117–126.

[115] Enterline and Henderson, "Type of Asbestos and Respiratory Cancer in the Asbestos Industry," p. 316.

[116] Henderson and Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," p. 121.

[117] Ibid.,Table 1, p. 118. The all-cause SMR was 120.4, and the SMR for cancer of the digestive tract was 137.8. There were 31 observed cases of pneumoconiosis and pulmonary fibrosis in the cohort, of which 19 were due to asbestosis.

[118] Ibid., pp. 124–125. The authors referred to a report by M. Borow et al. from hospitals in the vicinity of one of the plants studied by Henderson and Enterline, that of Manville, New Jersey. Borow reported 72 cases of mesothelioma from hospitals in the vicinity of this plant. A substantial number of these cases died prior to age 65, leading Henderson and Enterline to suggest that a feature in malignant mesothelioma is that death occurs at a relatively early age. See also, M. Borow et al., "Mesothelioma Following Exposure to Asbestos: A Review of 72 Cases," *Chest* 64 (November 1973): 641–646.

Focusing on the cement workers, for those working with chrysotile alone the SMR for lung cancer was 230.8 with a mean exposure of 255 mppcf-yrs; for those working with crocidolite and chrysotile in the cement pipe operations the SMR for lung cancer was very much higher at 521.7 with a mean exposure of 230 mppcf-yrs. There were 11 deaths from asbestosis among the cement shingle and sheet workers and only 2 among the cement pipe workers.[119]

(c) Scarborough, Ontario

We have already described in general terms, in Chapter 3, the disastrous health experience of the asbestos-cement workers at the Johns-Manville plant at Scarborough, Ontario, as reported by Dr. Murray M. Finkelstein. It will be recalled that this factory began production in 1948 manufacturing asbestos-cement pipe in one building and rock wool insulation in another. In 1955, production of asbestos-cement board commenced in a third building, and in 1960, the manufacture of asbestos insulation material was added.[120] The manufacture of asbestos-cement pipe involved both chrysotile and crocidolite, with crocidolite constituting approximately 20% of the asbestos used. As at New Orleans, it would appear that crocidolite constituted approximately 3% of the final product. In the manufacture of asbestos-cement board only chrysotile was used and this product was no longer made after 1970. No asbestos was used in the manufacture of rock wool insulation and these workers served as an internal control group.

Exposure information from the plant was scanty. Ontario Ministry of Labour hygienists performed surveys in 1949, 1956, 1961, 1968, and during the 1970s. Impinger surveys were performed for Johns-Manville by insurance company hygienists in 1954 and 1957, and Johns-Manville itself began annual measurements in 1961. However, there were no fibre counts until 1969, and reliable membrane filter sampling results from the government were not available until 1973.[121] There were two major ventilation changes in the pipe plant, the first, according to Dr. Finkelstein, having taken place in 1962 and the second in 1970.[122]

Based on the available data, Dr. Finkelstein made the following assumptions: (i) the relative dustiness of the jobs was unchanged between

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[119] Henderson and Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," pp. 121–123.

[120] See Chapter 3, Section A.3 of this Report.

[121] Murray M. Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," *American Review of Respiratory Disease* 125 (1982): 497. See also, RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 46–48.

[122] Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 497.

1948 and 1970; (ii) between 1955 and 1962, exposures were 30% higher than those in 1969–1970; (iii) exposures between 1948 and 1954 were twice as high as exposures in 1969–1970; and (iv) from 1962 to 1970, exposures were unchanged. Dr. Finkelstein constructed an exposure-response model using these assumptions in conjunction with the pre-ventilation change 1969–1970 fibre counts.[123]

We have already observed that Dr. Finkelstein conducted two separate analyses of the mortality experience of these workers: the first restricted to those 339 workers hired prior to 1960 and with at least 9 years' employment followed to October 1980;[124] the second extended to a cohort of 740 men employed one year or more, hired prior to 1960 and followed to the end of 1981.[125]

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[123] Ibid. See also, Murray M. Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," *British Journal of Industrial Medicine* 40 (1983): 139.

[124] Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 139. Of the 339 workers, 11 could not be classified from their work histories and were excluded from the analysis. Of the remaining 328 workers, 186 were production workers, 55 were maintenance workers, and 87 were employed in the rock wool/fibreglass operations or were otherwise minimally exposed to asbestos dust and thus constituted the control group.

[125] Murray M. Finkelstein, "Mortality in Asbestos-Cement Factory Workers," paper presented at the Second International Symposium on Epidemiology in Occupational Health, Montreal, 25 August 1982, pp. 1–2. (Mimeographed.) See also, Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983. (Mimeographed.) The composition of the cohort was as follows:

Numbers of Men at Risk in the Cohort and the Numbers Withdrawn from the Analysis at the End of 1977

	Years From Hire		Number Withdrawn	Percent Withdrawn
	10	20		
Production Workers	428	401	63	16%
Maintenance Workers	107	99	13	13%
Factory Controls	205	185	32	17%
Total	740	685	108	16%

SOURCE: Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983, Table 1, excerpt. (Mimeographed.)

The difference in numbers at the two dates from hire is due to death or emigration in the interval. All production workers were exposed to asbestos for at least one month prior to the end of 1961. Exposure-response calculations were made for the 401 production workers alive 20 or more years from their date of hire.

In the more restricted cohort of longer-term employees, among those with an average 18 years' cumulative exposure roughly estimated to be 180 f/cc-yrs, he observed a mesothelioma mortality rate of approximately 12 per thousand and an equal rate for lung cancer.[126] Even setting aside the exposure estimates, a striking statistic from this cohort is that, of the 58 deaths among production workers, 20 were caused by lung cancer and 10 more by mesothelioma. Table 5.13 shows mortality among the longer-term Scarborough factory workers, by selected causes of death, compared with the mortality predicted from Ontario population rates.

In Dr. Finkelstein's larger cohort of workers employed one year or more, the disease incidence has remained extraordinarily high. An indication of this fact can be gained from the mortality data for production workers 20 or more years from first employment: the SMR for all causes of death was 181; for lung cancer, 512; for gastrointestinal cancer, 285; and for non-malignant respiratory disease, 320 — figures which are clearly of the order of magnitude found by Dr. Dement and Dr. A.D. McDonald at Charleston, but which are considerably higher than those reported by Dr. Weill in New Orleans. Table 5.14 details observed and expected mortality for Dr. Finkelstein's larger cohort.

Dr. Finkelstein has also calculated 18-year cumulative exposures for the production workers in his extended cohort by combining work histories with the exposure estimates. Table 5.15 shows standardized mortality rates for these workers, for the period 20 or more years from first exposure, in relation to various exposure categories.

Of the 21 mesothelioma deaths which had already occurred in this cohort, 12 were pleural and 9 peritoneal, and it would appear that all but one had exposure to crocidolite in the pipe plant. Mesothelioma mortality rates increased significantly with time from first exposure.[127]

Dr. Finkelstein's study has been criticized for the uncertainty of the exposure information and for the fact that a clear dose-response relationship was not demonstrated for lung cancer. Dr. Finkelstein, in his testimony before the Commission, freely recognized these difficulties, but, as he also pointed out, his data do not compare unfavourably with those available in

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[126] Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 139. The mortality rate for lung cancer for Ontario men per 1,000 man-years is 1.6.

[127] Finkelstein, "Mortality in Asbestos-Cement Factory Workers," pp. 4-5. In this paper, Dr. Finkelstein indicated that 2 of the mesotheliomas occurred in the control group, but that one of these men had later moved to the Transite pipe section of the plant. The other man was not recorded as having worked in the asbestos areas. However, Dr. Finkelstein did observe that it was not uncommon for men from other areas of the plant to do weekend cleanups in the pipe shop, and these men may have been exposed in that fashion.

Table 5.13

Mortality Among the Factory Workers Compared with the Population of Ontario

Cause	Group*	Years Since First Exposure											
		15-19			20-24			25-33			Total: 20-33		
		Obs	Exp	O/E	Obs	Exp	O/E	Obs	Exp	O/E	Obs	Exp	O/E
All causes	P	8	8.9	1	16	11.8	1.4	34	11.6	2.9	50	23.4	2.1
	P + M	11	11.9	1	22	15.5	1.4	39	15.1	2.6	61	30.6	2.0
	C	7	5.0	1.4	7	5.9	1.2	7	7.4	1	14	13.3	1
All malignancies	P	2	1.9	1	9	2.8	3.2	20	2.9	6.9	29	5.7	5.1
ICD: 140-209	P + M	2	2.5	1	11	3.7	3.0	23	3.7	6.2	34	7.4	4.6
	C	3	1.1	2.7	3	1.4	2.1	1	1.8	1	4	3.2	1
Lung cancer	P	1	0.6	1	6	1.0	6.0	11	1.0	11.0	17	2.0	8.5
ICD: 162	P + M	1	0.8	1	7	1.2	5.8	12	1.3	9.2	19	2.5	7.6
	C	0	0.3	0	0	0.5	0	1	0.6	1	1	1.1	1
Mesothelioma	P	1	—	—	2	—	—	4	—	—	6	—	—
ICD: 163, 158, 228													
Gastrointestinal cancer	P	0	0.5	0	1	0.7	1	2	0.7	2.9	3	1.4	2.1
ICD: 150-154	P + M	0	0.7	0	1	0.9	1	3	0.9	3.3	4	1.8	2.2
	C	1	0.3	1	1	0.3	1	0	0.4	0	1	0.7	1
Non-malignant	P	1	0.4	1	1	0.7	1	3	0.8	3.8	4	1.5	2.7
respiratory disease	P + M	1	0.6	1	3	0.9	3.3	4	1.0	4.0	7	1.9	3.7
ICD: 460-519	C	0	0.3	0	0	0.4	0	1	0.5	1	1	0.9	1
Ischemic	P	4	3.9	1	2	4.7	0.4	5	4.6	1	7	9.3	0.8
heart disease	P + M	7	4.9	1.4	3	6.2	0.5	6	6.0	1	9	12.2	0.7
ICD: 410-414	C	3	2.1	1	1	2.4	0.4	2	2.9	1	3	5.3	0.6

Notes: *P means production workers. M means maintenance workers. C means factory controls (unexposed workers).

SOURCE: Murray M. Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," *British Journal of Industrial Medicine* 40 (1983): 140 (Table 2).

Table 5.14

Mortality Among the Factory Workers Compared
with the General Population of Ontario

		Years Since First Exposure											
		10 - 14		15 - 19		20 - 24		25 - 29		30 - 34		Total: 20 - 34	
Cause	Group*	O	SMR	O	SMR	O	SMR	O	SMR	O	SMR	O	SMR
All causes	P	6	50	17	96	26	113	50	272	10	161	86	181
	M	2	49	5	84	9	134	10	164	3	163	22	150
	C	4	56	12	123	12	102	9	78	7	130	28	98
All malignancies	P	0(1)**	[2.2]***	3(4)	79	14(17)	255	25(28)	543	5	333	44(50)	379
ICD: 140-209	M	1	125	1	79	2	124	7	461	3	638	12	333
(8th Revision)	C	1(2)	71	3	144	4(6)	191	2	71	3	234	9(11)	131
Lung cancer	P	0	[0.6]	2(3)	167	7(7)	368	13(13)	813	1	167	21(21)	512
ICD: 162	M	1	476	1	263	1	182	2	370	2	1,180	5	413
	C	0(1)	[0.4]	0	[0.7]	0(1)	[0.9]	1	103	2(1)	476	3(3)	130
Mesothelioma ICD: 228,	P	0(1)	= 0.4 per 10^3	1	= 0.4 per 10^3	2(5)	= 2.7 per 10^3	4(7)	= 6.3 per 10^3	2(3)	= 9.6 per 10^3	8(15)	= 4.4 per 10^3
158,163	M	0		0		1		1		0		2(2)	
	C	0		0		0		0(1)		0(1)		0(2)	
Gastrointestinal	P	0	[0.6]	0	[0.9]	3(2)	231	3(5)	273	2(1)	500	8(8)	285
cancer	M	0	[0.2]	0	[0.3]	0	[0.4]	1	278	0	[0.1]	1	118
ICD: 150-154	C	0	[0.4]	1	192	0(1)	[0.7]	1(0)	152	0	[0.3]	1(1)	61

Table 5.14 (continued)

Mortality Among the Factory Workers Compared with the General Population of Ontario

		Years Since First Exposure											
		10 - 14		15 - 19		20 - 24		25 - 29		30 - 34		Total: 20 - 34	
Cause	Group*	O	SMR	O	SMR	O	SMR	O	SMR	O	SMR	O	SMR
Non-malignant respiratory	P	0	[0.4]	1	122	2(1)	154	7(6)	636	0(1)	[0.4]	9(8)	320
diseases	M	0	[0.2]	0	[0.3]	3	789	1	256	0	[0.1]	4	444
ICD: 460-519	C	0	[0.3]	0	[0.5]	0	[0.7]	1	130	0	[0.4]	1	54
Ischemic	P	4	86	7	98	2(1)	22	6	82	3	120	11(10)	58
heart disease	M	0	[1.7]	4	163	2	74	1	41	0	[0.7]	3	51
ICD: 410-414	C	1	35	4	100	5(4)	106	4	89	3	141	12(11)	106

Notes: *P means production workers. M means maintenance workers. C means factory controls (unexposed workers).

**Numbers in parentheses reflect "best evidence" classification.

***Numbers in brackets are numbers of deaths expected.

SOURCE: Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983, Table 2. (Mimeographed.)

Table 5.15

Mortality Rates in Relation to Cumulative Exposure

Cause of Death	Mortality Rates per 10^3 Man-Years*							Test for Trend
	Ontario Males**	Factory Controls	≤ 30 f/cc-yrs	30.1-75 f/cc-yrs	75.1-105 f/cc-yrs	105.1-150 f/cc-yrs	> 150 f/cc-yrs	
Lung cancer	1.3	1.7 (3)†	3.0 (3)	8.0 (6)	15.7 (5)	11.7 (5)	3.5 (2)	$Z = 2.5$; $P < 0.01$.
Mesothelioma	$\ll 1$	1.1 (2)	4.1 (4)	3.8 (3)	2.4 (1)	2.7 (1)	17.9 (6)	$Z = 3.5$; $P < 0.01$.
Gastrointestinal cancer	1.0	0	2.0 (2)	3.5 (2)	2.4 (1)	0	10.1 (3)	$Z = 2.3$; $P < 0.02$.
All asbestos cancer	2.3	2.8 (5)	9.1 (9)	15.4 (11)	20.6 (7)	14.4 (6)	31.5 (11)	
Non-malignant respiratory disease	1.2	0.6 (1)	2.0 (2)	1.4 (1)	4.9 (2)	6.2 (2)	0.9 (1)	$Z = 1.6$; $0.05 < P < 0.06$.
Ischemic heart disease	7.4***	6.4 (11)	1.0 (1)	1.5 (1)	6.7 (3)	17.8 (4)	2.4 (1)	$Z = 0.3$; $P > 0.3$.
All causes of death	17.9	16.2	20.1	26.7	40.2	39.6	41.4	$Z = 4.4$; $P < 0.01$.
Number of men in exposure category at 20 years		185	169	113	37	43	39	
Total number of deaths 20 - 34 years		28	20	21	15	13	17	

Notes: *Standardized to the age and latency distribution of the cohort as a whole, for the period beyond 20 years from first exposure.

**From Ontario Vital Statistics 1970-1974.

***From Ontario Vital Statistics 1969-1973.

†Numbers in parentheses are the actual numbers of deaths.

SOURCE: Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983, Table 6. (Mimeographed.)

many other studies. Moreover, he estimates that his calculations of cumulative exposure are accurate to within a factor of 3 to 5.[128] Notwithstanding the potential for error arising out of the uncertainty of the exposure information, the disease experience at Scarborough is remarkably high.

(d) Cardiff, Wales

H.F. Thomas et al. of the Medical Research Council Epidemiology Unit at Cardiff, Wales, have recently reported on the mortality experience of 1,970 workers employed at an asbestos-cement factory near Cardiff for at least 6 months between 1936 and 1977.[129] Crocidolite was used at this factory between 1932 and 1935, but from 1935 onwards until the factory closed in 1980 only chrysotile from Canada and South Africa was used, principally for the manufacture of cement sheeting and associated fittings for the building industry. Over 95% of the cohort was traced by the end of 1977 and 381 deaths were identified.

Detailed analysis was restricted to the 1,592 male employees among whom there were 351 observed deaths. These male workers were separated into three categories: all male workers; those alive 15 or more years after first exposure; and those employed in 1935–1936 and thus potentially exposed to crocidolite. In none of the three groups was there any apparent increase in general mortality, cancer mortality, or in mortality specifically from lung cancer and gastrointestinal cancer. Table 5.16 shows the actual standard mortality ratios for selected causes of death. There were 2 cases of pleural mesothelioma in the cohort, and both were employed at the factory when crocidolite was used.[130] Thus, the general results of the study suggest that the chrysotile cement workers are not at any increased risk from asbestos exposure. This conclusion, however, must be treated with some caution. There was no minimum latency period for the entire cohort. Further, over half the cohort studied was employed less than 2 years, and three-quarters worked in the factory for less than 4 years. No individual exposure data have been presented for the cohort, although the authors commented that dust levels in asbestos-cement factories are generally lower than in other asbestos-handling industries, and in the Cardiff plant there was a pronounced improvement in dust levels from the mid-1960s onwards. The authors concluded their published study by commenting that while it "... gives reassuring findings on the long-term mortality experience of an

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[128] The issue of Dr. Finkelstein's exposure estimate is discussed in greater detail in Chapter 3, Section A.3.

[129] H.F. Thomas et al., "Further Follow-up Study of Workers from an Asbestos Cement Factory," *British Journal of Industrial Medicine* 39:3 (August 1982): 273–276.

[130] Ibid., pp. 274–275.

Table 5.16

Mortality by All Causes, All Neoplasms, and Selected Cancers for the Total Male Population, Men Alive 15 Years or Longer After First Exposure, and for Men Employed in 1935-1936

Cause of Death (ICD Code)	Study Group	Expected Deaths	Observed Deaths	Standardized Mortality Ratio (SMR)
All causes	Total population	343.3	351	102
	Alive ≥ 15 years after first exposure	243.2	261	107
	Employed in 1935-1936	88.3	83	94
All neoplasms (140-239)	Total population	80.7	74	92
	Alive ≥ 15 years after first exposure	61.0	58	95
	Employed in 1935-1936	21.5	22	103
Cancers of lung and pleura (162, 163)	Total population	33.0	30	93
	Alive ≥ 15 years after first exposure	25.8	24	93
	Employed in 1935-1936	9.2	7	76
Cancers of gastrointestinal tract (151-154)	Total population	19.6	18	92
	Alive ≥ 15 years after first exposure	14.1	14	99
	Employed in 1935-1936	5.0	6	120

SOURCE: H.F. Thomas et al., "Further Follow-up Study of Workers from an Asbestos Cement Factory," *British Journal of Industrial Medicine* 39:3 (August 1982): 274 (Table 2).

asbestos cement factory population, the high labour turnover does not enable conclusions to be drawn on the hazards of sustained long-term exposure to chrysotile asbestos."[131]

B.5 Asbestos Manufacturing — Amosite Insulation Products

Paterson, New Jersey

Just prior to the entry of the United States into World War II, the Union Asbestos and Rubber Company (UNARCO) established an amosite asbestos factory at Paterson, New Jersey, to supply the U.S. Navy with asbestos insulation for the pipes and boilers of its ships. From June 1941 until December 1945, 933 men were hired to work in the plant which continued in operation until 1954. The health experience of these workers was reported on by Dr. Herbert Seidman and his colleagues at the Mount Sinai School of Medicine in New York in 1979.[132]

As the employees of Paterson were among those not conscripted for war service, they tended to be considerably older in age than a peace-time workforce. Most worked for short periods of time, many for less than 9 months, and only a small number worked longer than 2 years. Following the closing of the Paterson plant, few returned to employment with asbestos.[133] There were no direct measurements of dust levels in the plant; however, measurements made more recently in two other plants operated by UNARCO — one in Tyler, Texas, and the other in Port Allegany, Pennsylvania — making the same product, from the same fibre type (amosite), to the same specifications, and using the same type of machinery, were extremely high, averaging 34.9 f/cc.[134]

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[131] Ibid., pp. 275-276.

[132] Herbert Seidman, Irving J. Selikoff, and E. Cuyler Hammond, "Short-Term Asbestos Work Exposure and Long-Term Observation," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 61-89. There is an earlier report of the same cohort by Herbert Seidman, Ruth Lilis, and Irving J. Selikoff, "Short-Term Asbestos Exposure and Delayed Cancer Risk," in *Prevention and Detection of Cancer,* vol. 1: *Etiology,* ed. H.E. Nieburgs (New York: Marcel Dekker, Inc., 1977), pp. 943-960.

[133] Seidman, Selikoff, and Hammond, "Short-Term Asbestos Work Exposure and Long-Term Observation," p. 62.

[134] In their published article, the authors suggested dust counts as high as 23 f/cc. The 34.9 f/cc figure comes from William J. Nicholson, "Criteria Document for Swedish Occupational Standards: Asbestos and Inorganic Fibers," *Arbete Och Hälsa* 17 (1981): 22-23. The figure was based upon measurements made by the U.S. Public Health Service during 1967, 1970, and 1971. The arithmetic averages of these exposure measurements for Tyler and for Port Allegany were 39.1 and 28.9 f/cc respectively, yielding an overall average of 34.9 f/cc.

The health experience at Paterson therefore reflects both the unusual characteristics of the workforce and the rather unusual nature of their asbestos exposure — short but very intense exposure (to amosite) — followed by a long period of observation, 35 or more years from first exposure. In order to investigate only the health effects of asbestos exposure at Paterson, Dr. Seidman excluded for the purpose of analysis 113 employees who had asbestos exposure elsewhere, leaving 820 in the cohort.

By the time he reported, 528 workers in the cohort had died, 93 from lung cancer and 14 from mesothelioma, and the workers employed 2 or more years were close to extinction (157 deaths out of 188 workers).[135] Lung cancer mortality rates were extremely high, with SMRs ranging from 294 for those with less than one month's employment to 650 for those employed more than 2 years.[136] Many were already at or near "cancer ages" when hired and died within a relatively short period of time, 5 to 14 years after onset of work.[137]

Based on the health experience at Paterson, which is admittedly marked by unusual circumstances, working with amosite asbestos in the manufacture of insulation products for just one year at 1 f/cc would yield an excess risk of lung cancer of 9.1%. Working for 10 years at 1 f/cc would double that risk.[138]

B.6 Asbestos Outside Fixed Place Industry

North American Insulators

Commencing in the 1960s, Dr. Irving J. Selikoff and his associates at the Mount Sinai School of Medicine in New York have been following the mortality experience of a large group of insulation workers in the United

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[135] Seidman, Selikoff, and Hammond, "Short-Term Asbestos Work Exposure and Long-Term Observation," pp. 66–67.

[136] Nicholson, "Criteria Document for Swedish Occupational Standards: Asbestos and Inorganic Fibers," p. 21.

[137] Seidman, Selikoff, and Hammond, "Short-Term Asbestos Work Exposure and Long-Term Observation," pp. 87–88.

[138] RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 65.

States and Canada.[139] These workers were members of the International Association of Heat and Frost Insulators and Asbestos Workers. They have been primarily employed in the building trades (engaged in doing construction insulation work), but have also worked in refineries, shipyards, and on repair jobs. These insulators have generally been active in all parts of the trade with conditions varying from job to job and from company to company. According to Dr. Selikoff, fewer than 10% have remained with one company during their working lives.[140]

Until approximately the early 1930s, chrysotile alone was utilized in the manufacture of the insulation products used by these men. Amosite began to be used in the mid-1930s in small quantities but became more extensively utilized during and following World War II. Few dust counts were made in insulation work until the 1960s, and Dr. Selikoff has presented no exposure data in his studies. He has, however, suggested, based on reconstruction of work situations and extrapolation to the past of observations made more recently, that these insulation workers would have been exposed to dust levels of between 4 and 12 f/cc. While there may have been work periods of little or no exposure, there were also likely times of peak exposures much higher than the calculated averages.[141]

Dr. Selikoff has conducted mortality analyses on two cohorts of insulators. The first was a small group of 632 workers who, as of January 1, 1943, were registered as members of the union in the New York - New Jersey metropolitan area. By the end of 1976, 478 of the original 632 workers had died. Excess mortality due to asbestos-related diseases was striking. Of the total deaths in the cohort, 93 were from lung cancer, 38 from mesothelioma (11 pleural and 27 peritoneal), and 41 from asbestosis (of which 38 were in the period 35 or more years from first exposure). The SMRs for lung cancer, cancer of the stomach, and cancer of the colon or rectum were respectively 699, 352, and 277.[142]

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[139] Irving J. Selikoff, Jacob Churg, and E. Cuyler Hammond, "Asbestos Exposure and Neoplasia," *Journal of the American Medical Association* 188 (1964): 22-26; Irving J. Selikoff, Jacob Churg, and E. Cuyler Hammond, "The Occurrence of Asbestosis Among Insulation Workers in the United States," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 139-155; Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Cancer Risk of Insulation Workers in the United States," in *Biological Effects of Asbestos*, eds. P. Bogovski et al., IARC Scientific Publications, no. 8 (Lyon, France: International Agency for Research on Cancer, 1973), pp. 209-216; and Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 91-116.

[140] Selikoff, Hammond, and Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," pp. 91-92.

[141] Ibid., pp. 92-93.

[142] Ibid., pp. 93-95.

The second cohort investigated by Dr. Selikoff was very much larger and comprised all 17,800 workers on the union rolls both in Canada and the United States as of January 1, 1967. Observation of this group was maintained through to the end of 1976, and in that 9-year period there were 2,271 deaths. According to the best available evidence, 175 of these deaths were from mesothelioma (63 pleural and 112 peritoneal), 168 from asbestosis, and 486 from lung cancer. Altogether these three causes of death were responsible for approximately 37% of all deaths in the cohort. The SMRs for lung cancer, gastrointestinal cancer, and cancer of the larynx were respectively 460, 167, and 234. (See Table 5.17.) The authors of the study justifiably concluded that these asbestos insulation workers suffered an extraordinarily increased risk of death from cancer and from asbestosis associated with their employment. It should be noted that little of the excess mortality was observed in the period less than 15 years from first exposure and most deaths occurred two, three, and four or more decades from the onset of exposure.[143]

B.7 Summary

This review of the epidemiological evidence, which is summarized in Table 5.18, has served to illustrate that there does appear to be an association between the incidence of asbestos-related disease and the type of industrial process in which asbestos is used. The health risk in chrysotile friction products manufacturing and chrysotile mining and milling pales by comparison to the health risk in chrysotile textile production. Insulators seem to be at greater risk than miners and millers and most manufacturing workers.

And yet, the differences in industrial processes do not account for all of the observed differences in the results of the various epidemiological studies. Even among plants using similar processes and making similar products there has been a wide divergence in the incidence of disease. The mortality experience of the asbestos-cement workers at Scarborough ranks on a par with that of the textile workers at Charleston and has been considerably worse than the experience of the New Orleans workforce. In turn, the cement workers in New Orleans and among Dr. Enterline's Manville employees have fared worse than the chrysotile cement workers at Cardiff. The overall incidence of asbestos-related disease at Rochdale, while not insignificant and roughly similar to that at Lancaster, has been less than that experienced at Charleston. The incidence of mesothelioma in the mines of Western Australia and of the Cape region in South Africa has been markedly different than its incidence in the Quebec mines or even apparently in the mining region of the Transvaal. The number of mesotheliomas at

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[143] Ibid., pp. 106–110.

Table 5.17

Deaths Among 17,800 Asbestos Insulation Workers in the United States and Canada, January 1, 1967 - December 31, 1976 (Number of Men: 17,800; Man-Years of Observation: 166,853)

Underlying Cause of Death	Expected*	Observed (BE)	Observed (DC)	Ratio O/E (BE)	Ratio O/E (DC)
Total deaths, all causes	1,658.9	2,271	2,271	1.37	1.37
Total cancer, all sites	319.7	995	922	3.11	2.88
Cancer of lung	105.6	486	429	4.60	4.06
Pleural mesothelioma	**	63	25	—	—
Peritoneal mesothelioma	**	112	24	—	—
Mesothelioma, not otherwise stated	**	0	55	—	—
Cancer of esophagus	7.1	18	18	2.53	2.53
Cancer of stomach	14.2	22	18	1.54	1.26
Cancer of colon-rectum	38.1	59	58	1.55	1.52
Cancer of larynx	4.7	11	9	2.34	1.91
Cancer of pharynx, buccal	10.1	21	16	2.08	1.59
Cancer of kidney	8.1	19	18	2.36	2.23
All other cancer	131.8	184	252	1.40	1.91
Non-infectious pulmonary diseases, total	59.0	212	188	3.59	3.19
Asbestosis	**	168	78	—	—
All other causes	1,280.2	1,064	1,161	0.83	0.91

Notes: *Expected deaths are based upon white male age-specific U.S. death rates of the U.S. National Center for Health Statistics, 1967-1976.

**Rates are not available, but these have been rare causes of death in the general population.

(BE): Best evidence. Number of deaths categorized after review of best available information (autopsy, surgical, clinical).

(DC): Number of deaths as recorded from death certificate information only.

SOURCE: Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 103 (Table 12).

Table 5.18

Mortality from Asbestos-Related Diseases in Various Cohort Studies

Type of Activity	Study	Place	Fibre Type
Mining	J.C. McDonald, 1980	Quebec	Chrysotile
	Nicholson, 1979	Quebec	Chrysotile
	Rubino, 1979	Italy	Chrysotile
	Hobbs, 1980	Western Australia	Crocidolite
	Meurman, 1974	Finland	Anthophyllite
Friction Materials	Berry, 1983	England	Chrysotile, Crocidolite[m]
	A.D. McDonald, 1984	Connecticut	Chrysotile, Crocidolite[m], Anthophyllite[m]
Textiles	Peto, 1977	England	Chrysotile, Crocidolite (?)
	Peto, 1980	England	Chrysotile, Crocidolite (?)
	Dement, 1982	South Carolina	Chrysotile, Crocidolite[m]
	A.D. McDonald, 1983	South Carolina	Chrysotile, Crocidolite[m]
	A.D. McDonald, 1983	Pennsylvania	Chrysotile, Amosite, Crocidolite[m]
	Robinson, 1979	Pennsylvania	Chrysotile, Amosite[m], Crocidolite[m]
Cement Products	Weill, 1979	New Orleans	Chrysotile, Crocidolite, Amosite[m]
	Finkelstein, 1983	Scarborough	Chrysotile, Crocidolite
	Thomas, 1982	Cardiff	Chrysotile, Crocidolite[m]
General Manufacturing	Henderson and Enterline, 1979	United States	Chrysotile, Crocidolite, Amosite
	Newhouse, 1979	England	Chrysotile, Crocidolite, Amosite
Gas Mask Manufacturing	Jones, 1980	England	Crocidolite
Insulation Products	Seidman, 1979	New Jersey	Amosite
Insulators	Newhouse, 1979	England	Chrysotile, Crocidolite, Amosite
	Selikoff, 1979	United States and Canada	Chrysotile, Amosite
	Selikoff, 1979	New York — New Jersey	Chrysotile, Amosite
Shipyard Workers	Rossiter, 1980	England	Chrysotile, Crocidolite, Amosite

	Number in Cohort	Total Deaths	Meso-thelioma	Asbestosis (Pneumo-coniosis)	Lung Cancer			Gastrointestinal Cancer[a]		
					Obs	Exp	SMR	Obs	Exp	SMR
	10,939	3,291	10	42	230	184	125	209	203	103
	544	178	1	26	28	11.1	252	10	9.5	105
	952	332	0	9	11[b]	10.4	106	19	19.3	98
	6,200	526	17[c]	14	60	38.2	157	NS		
	1,092	248	0	13	21	12.6	167	7	14.9	47
M	9,113	1,640	8	NS	143[d]	139.5	103	103	107.2	96
F	4,347	346	2	NS	6[d]	11.3	53	29	27.4	106
	3,641	1,267	0	12[e]	73[f]	49.1	148.7	59[f]	51.6	114.4
	1,106	317	10	NS	51[d]	23.8	214	16	15.7	102
	679	239	7	10	40	23.3	172	NS		
	768	191	1	15	26	7.5	348	9	7.1	127
	2,543	857	1	21	59[f]	29.6	199.5	26[f]	17.1	151.7
	4,137	1,392	14	74	53[f]	50.5	105	54[f]	47.9	112.7
M	2,722	912	13	NS	49	36.1	136	50	41.4	121
F	554	128	4	NS	14	1.7	824	8	6.0	133
	5,645	601	0[g]	NS	51	49.2	104	25	50.1	50
	535	138	19	NS	26[f]	5.4	480	9[f]	3.8	240
	1,592	351	2	NS	28	33.0	85	18	19.6	92
	1,075	781	5	31	63[h]	23.3	270.4	55	39.9	137.8
M	2,887	545	46	NS	103[d]	43.2	238	40[i]	34	118
F	693	200	21	NS	27[d]	3.2	844	20[i]	10.2	196
	578	166	17	NS	12	6.3	190	10	20.3	49
	820	528	14[j]	30[j]	93[j]	22.8	408	NS		
	1,368	83	10	NS	21[d]	5.6	375	3[i]	4.3	70
	17,800	2,271	175[j]	168[j]	486[j]	105.6	460	99[j]	59.4	167
	632	478	38[j]	41[j]	93	13.3	699	43	15.1	285
	6,076	1,043	31	9	84	119.7	70	63	83.3	76

Table 5.18 (continued)

Mortality from Asbestos-Related Diseases in Various Cohort Studies

Notes:

[a] The figures in this table for "Gastrointestinal Cancer" may not be entirely comparable among the various cohort studies since some studies give figures for all abdominal or digestive cancers and others for gastrointestinal cancer only. The figures used in the actual studies have been reproduced in this table.

[b] Includes one suspected case of mesothelioma.

[c] According to the mortality study, which was restricted to deaths before January 1, 1978. The text of this study also noted 26 cases of mesothelioma diagnosed to January 1, 1979.

[d] Pleural mesotheliomas included in lung cancer total given by the authors but taken out of the lung cancer total for the purpose of this table.

[e] Authors stated that none of the cases were clearly attributable to asbestos exposure.

[f] Number of deaths 20 or more years from first exposure.

[g] Two cases not meeting criteria for entry into the cohort.

[h] Includes mesotheliomas.

[i] Peritoneal mesotheliomas included in gastrointestinal cancer total given by the authors but taken out of the gastrointestinal cancer total for the purpose of this table.

[j] "Best Evidence" figure.

[m] Minimal usage.

NS means not stated.
M means male.
F means female.

SOURCES:	J.C. McDonald, 1980:	J. Corbett McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," *British Journal of Industrial Medicine* 37 (1980): 11-24.
	Nicholson, 1979:	William J. Nicholson et al., "Long-Term Mortality Experience of Chrysotile Miners and Millers in Thetford Mines, Quebec," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 11-21.
	Rubino, 1979:	G.F. Rubino et al., "Mortality of Chrysotile Asbestos Workers at the Balangero Mine, Northern Italy," *British Journal of Industrial Medicine* 36 (1979): 187-194.
	Hobbs, 1980:	M.S.T. Hobbs et al., "The Incidence of Pneumoconiosis, Mesothelioma and Other Respiratory Cancer in Men Engaged in Mining and Milling Crocidolite in Western Australia," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 615-625.
	Meurman, 1974:	L.O. Meurman, R. Kiviluoto, and M. Hakama, "Mortality and Morbidity Among the Working Population of Anthophyllite Asbestos Miners in Finland," *British Journal of Industrial Medicine* 31:2 (April 1974): 105-112.
	Berry, 1983:	Geoffrey Berry and Muriel L. Newhouse, "Mortality of Workers Manufacturing Friction Materials Using

SOURCES: (continued)

Asbestos," *British Journal of Industrial Medicine* 40:1 (February 1983): 1-7. See also, Muriel L. Newhouse, Geoffrey Berry, and Joseph W. Skidmore, "A Mortality Study of Workers Manufacturing Friction Materials with Chrysotile Asbestos," *Annals of Occupational Hygiene* 26:1-4 (1982): 899-909.

A.D. McDonald, 1984: Alison D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant," *British Journal of Industrial Medicine*, in press, 1984.

Peto, 1977: Julian Peto et al., "A Mortality Study Among Workers in an English Asbestos Factory," *British Journal of Industrial Medicine* 34 (1977): 169-173.

Peto, 1980: Julian Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 829-836.

Dement, 1982: John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," *Annals of Occupational Hygiene* 26:1-4 (1982): 869-887.

A.D. McDonald, 1983: Alison D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," *British Journal of Industrial Medicine* 40 (1983): 361-367.

A.D. McDonald, 1983: Alison D. McDonald et al., "Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture," *British Journal of Industrial Medicine* 40 (1983): 368-374.

Robinson, 1979: Cynthia F. Robinson, Richard A. Lemen, and Joseph K. Wagoner, "Mortality Patterns, 1940-1975 Among Workers Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility," in *Dusts and Disease*, eds. Richard A. Lemen and John M. Dement (Park Forest South, Illinois: Pathatox Publishers, Inc., 1979), pp. 131-143.

Weill, 1979: Hans Weill, Janet Hughes, and Carmel Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," *American Review of Respiratory Disease* 120:2 (August 1979): 345-354.

Finkelstein, 1983: Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983. (Mimeographed.)

Thomas, 1982: H.F. Thomas et al., "Further Follow-up Study of Workers from an Asbestos Cement Factory," *British Journal of Industrial Medicine* 39:3 (August 1982): 273-276.

SOURCES: (continued)	Henderson and Enterline, 1979:	Vivian L. Henderson and Philip E. Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 117-126.
	Newhouse, 1979:	Muriel L. Newhouse and Geoffrey Berry, "Patterns of Mortality in Asbestos Factory Workers in London," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 53-60.
	Jones, 1980:	J.S.P. Jones et al., "The Consequences of Exposure to Asbestos Dust in a Wartime Gas-Mask Factory," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 637-653.
	Seidman, 1979:	Herbert Seidman, Irving J. Selikoff, and E. Cuyler Hammond, "Short-Term Asbestos Work Exposure and Long-Term Observation," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 61-89.
	Newhouse, 1979:	Muriel L. Newhouse and Geoffrey Berry, "Patterns of Mortality in Asbestos Factory Workers in London," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 53-60.
	Selikoff, 1979:	Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 91-116.
	Selikoff, 1979:	Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 91-116.
	Rossiter, 1980:	Charles E. Rossiter and Ruth M. Coles, "HM Dockyard, Devonport: 1947 Mortality Study," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 713-721.

Lancaster stands in stark contrast to the virtual absence of this disease at Charleston.

Thus, we conclude that while the health risks of asbestos are a function of industrial process, process alone does not provide a complete explanation of the incidence of disease that has been recorded. We turn, then, to a second factor that appears to have affected the incidence of asbestos-related disease: fibre type.

C. The Effect of Fibre Type

C.1 Introduction

The present Ontario Regulation Respecting Asbestos establishes control limits which clearly differentiate by fibre type.[144] In keeping with the recommendations of the U.K. Advisory Committee on Asbestos, the Ontario asbestos regulation reflects the view that of the three principal fibre types, chrysotile is the least hazardous and crocidolite the most hazardous, with amosite occupying an intermediate position. However, this view is one that has by no means gone unchallenged. There is perhaps no issue related to the health effects of asbestos that has evoked as much debate as the issue of whether the amphiboles, and particularly crocidolite, are more hazardous than chrysotile or whether they are all equally hazardous. At the time of our hearings, both the medical and the scientific communities, as well as regulatory agencies, appeared divided on this issue, and their division of opinion was quite evident during testimony before us. For example, in contrast to the position taken by the U.K. Advisory Committee,[145] Mr. Richard A. Lemen of NIOSH testified that the Institute as well as the United States regulatory agency, the Occupational Safety and Health Administration (OSHA), were of the opinion that the health evidence did not warrant any differentiation on the basis of fibre type.[146] Dr. William J. Nicholson and Mr. Julian Peto, among others, expressed similar views.[147] At the other end of the spectrum, Dr. J. Corbett McDonald and his associates were firmly of the view that amphibole exposure was considerably more hazardous than chrysotile exposure, and so much so that the amphiboles ought to be banned.[148]

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[144] Regulation Respecting Asbestos, O. Reg. 570/82, ss. 4(1) and 4(2), made under the *Occupational Health and Safety Act*, R.S.O. 1980, c. 321.

[145] U.K., Advisory Committee on Asbestos, *Asbestos — Volume I: Final Report of the Advisory Committee*, paragraphs 176–211, pp. 74–78.

[146] RCA Transcript, Evidence of Mr. Richard A. Lemen, 8 July 1981, Volume no. 17, pp. 12–14.

[147] RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 78–79; and RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 61–63, 126–127. See also, RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, pp. 47–48; and RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 26 August 1981, Volume no. 30, pp. 54–55.

We do not pretend that we have a definitive resolution to the scientific controversy over fibre type; our interest lies in whether the weight of the evidence supports differentiation for regulatory purposes. The evidence from various studies, both epidemiological and experimental, gives us a considerable amount of information about the relative hazards of chrysotile, crocidolite, and amosite. In order to sort out this evidence in a sensible manner, we believe it is helpful to consider the evidence for and against differentiation by fibre type in relation to each asbestos-related disease separately. The principal focus of our analysis is whether the amphiboles (crocidolite and amosite) are or are not more hazardous than chrysotile. We separately consider whether there is any evidentiary basis for differentiating between crocidolite and amosite. Finally, we consider briefly the evidence as to the health effects of anthophyllite, tremolite, and actinolite.

C.2 The Evidence in Relation to Asbestosis

The epidemiological evidence that might indicate a differentiation according to fibre type in relation to the development of asbestosis is in our judgement minimal. There does exist one study providing an internal comparison which suggests that crocidolite may have a greater fibrogenic effect than chrysotile. This is Dr. Weill's study,[149] the results of which were cited in the report of the U.K. Advisory Committee.[150] Dr. Weill examined the clinical, radiographic, and physiological indicators of lung disease among two groups of workers who had spent 20 to 30 years in the asbestos-cement industry in New Orleans. One group had exposure to chrysotile, silica, and crocidolite in the pipe-making area. The other group had no crocidolite exposure at all, having worked only with chrysotile. Dr. Weill found that the crocidolite-exposed group had a significantly increased prevalence of small irregular opacities and pleural thickening, smaller lung volumes, lower forced expiratory flow, reduced pulmonary diffusion, and a greater prevalence of clubbing than the group not exposed to crocidolite.[151] The radiographic characteristics of the two groups of workers are set out in Table 5.19.

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[148] RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 46–47; and 25 June 1981, Volume no. 13, p. 53.

[149] Hans Weill et al., "Lung Function Consequences of Dust Exposure in Asbestos Cement Manufacturing Plants," *Archives of Environmental Health* 30:2 (February 1975): 88–97. See also, Hans Weill et al., "Differences in Lung Effects Resulting from Chrysotile and Crocidolite Exposure," in *Inhaled Particles IV*, Part 2, ed. W.H. Walton (Oxford: Pergamon Press, 1977), pp. 789–798.

[150] Acheson and Gardner, "The Ill Effects of Asbestos on Health," paragraph 121, p. 26.

On the other hand, a recent report by Dr. Margaret R. Becklake and her colleagues of asbestos workers in two plants in Montreal — a textile plant using only chrysotile and a plant manufacturing insulation and cement products in which some amosite and crocidolite had been used along with chrysotile — failed to demonstrate that fibre type had a significant influence on the prevalence of respiratory symptoms and functional abnormalities.[152] Rather, the findings suggested that process, not fibre type, was the dominant factor affecting the results.[153] (See Table 5.20.) Similarly, in parallel investigations by Dr. A.D. McDonald et al. of United States asbestos factories, exposure-response relationships for asbestosis in the two textile plants studied — the one in Charleston and the other in Pennsylvania — were similar, notwithstanding that the former used only chrysotile while the latter used both crocidolite and amosite in addition to chrysotile.[154]

The epidemiological findings of Dr. Becklake and Dr. A.D. McDonald are supported by animal inhalation experiments. These experiments have not only demonstrated that all fibre types are fibrogenic, but have suggested that if anything chrysotile was not less but more fibrogenic than the amphiboles. For example, in 1974, Dr. J. Christopher Wagner and his associates reported the results of two experiments in which rats were exposed by inhalation to dust clouds of the UICC (Union Internationale Contre le Cancer) standard reference samples.[155] All samples produced

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[151] Weill et al., "Differences in Lung Effects Resulting from Chrysotile and Crocidolite Exposure," p. 789. In his evidence, Dr. Weill suggested that the greater fibrogenic effect attributable to crocidolite could have been due to the physical dimension of the fibre or could simply have been due to the dustiness of the fibre. See RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 36–37.

[152] Margaret R. Becklake et al., "Exposure to Asbestos and Respiratory Abnormality: The Influence of Fibre Type and Nature of Exposure," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 763–768.

[153] Ibid. The authors carried out health surveys on workers in two plants in the Montreal region: a textile plant in which only chrysotile had been used and a plant manufacturing insulation and cement products in which chrysotile was the dominant fibre used, but in which both amosite and crocidolite had also been used. The results were compared with results previously obtained in the chrysotile mining industry in Quebec. Despite apparently shorter exposure to lower dust levels, asbestos exposure in the manufacturing sector had more effect on respiratory status than exposure in mining and milling. Between-plant differences were less than between-process differences.

[154] A.D. McDonald et al., "Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture," p. 373.

[155] J. Christopher Wagner et al., "The Effects of the Inhalation of Asbestos in Rats," *British Journal of Cancer* 29:3 (March 1974): 252–269.

Table 5.19

Radiographic Characteristics

Radiographic Abnormality	75% or More in Pipe Area (108)	Never Worked in Pipe Area (100)	
Small rounded opacities			
1/1+	6 (5.6%)	4 (4%)	Non-significant
Small irregular opacities			
1/1+	11 (10.2%)	2 (2%)	P = 0.02
Pleural thickening			
1+	34 (31%)	15 (15%)	P = 0.006

SOURCE: Hans Weill et al., "Differences in Lung Effects Resulting from Chrysotile and Crocidolite Exposure," in *Inhaled Particles IV,* Part 2, ed. W.H. Walton (Oxford: Pergamon Press, 1977), Table 2, p. 792.

Table 5.20

Prevalence (%) of Abnormality, Adjusted for Age and Smoking Status, in Three Workforces Exposed to Asbestos

Radiographic Abnormality	Insulation and Cement Products	Textiles	Mining and Milling
Rounded opacities	1.5	1.6	1.8
Irregular opacities	19.7	20.9	6.6
Pleural thickening	20.8	15.7	4.7
Pleural calcification	3.4	2.5	3.4

SOURCE: Presented during Ontario, Royal Commission on Asbestos, Transcript of Public Hearings, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20. Revised during telephone communication between Dr. Margaret R. Becklake, Department of Epidemiology and Health, McGill University, Montreal, Quebec and Royal Commission on Asbestos Staff, 28 October 1983.

asbestosis which continued to progress after removal from exposure. There was no evidence of less asbestosis among the rats exposed to chrysotile than those exposed to the amphiboles even though much less dust was retained in the lungs of the chrysotile-exposed animals. (See Table 5.21.) In a more recent inhalation study by Dr. John M.G. Davis, the chrysotile dust clouds produced far more interstitial pulmonary fibrosis than the amphibole clouds when the animals were examined 29 months after the start of inhalation.[156] (See Table 5.22.) These results appear to us representative of the animal inhalation studies that have investigated the issue of fibre type in relation to asbestosis.[157]

C.3 The Evidence in Relation to Lung Cancer

When we turn to the incidence of lung cancer, there is more evidence than there is with respect to asbestosis. However, as we shall see, this evidence does not appear to us to yield any consistent pattern that, absent differences in industrial process or other factors, would implicate the amphiboles per se as more hazardous.

Perhaps the strongest evidence against the amphiboles comes from the experience of the factory and maintenance workers studied by Dr. Weill and by Dr. Enterline in their respective cohort studies. In order to assess the potential influence of fibre type on the risk of lung cancer, Dr. Weill, as we have already observed, compared the mortality experience of that part of his cohort exposed to chrysotile only (4,201 workers) with two groups of workers exposed to crocidolite as well: those with steady exposure in the pipe plant (1,004 workers) and those with intermittent exposure to crocidolite through occasional maintenance work in the area (235 workers). Follow-up and exposure data for these groups were roughly similar.[158] Dr.

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[156] John M.G. Davis, "The Use of Animal Inhalation Experiments in the Study of Asbestos Bioeffects," *Staub-Reinhalt. Luft* 40:10 (October 1980): 453–456.

[157] The animal evidence is reviewed in detail in U.S., National Institute for Occupational Safety and Health, *Revised Recommended Asbestos Standard*, prepared by Richard A. Lemen and John M. Dement (Washington, D.C.: U.S. Department of Health, Education and Welfare, December 1976); World Health Organization, International Agency for Research on Cancer Working Group, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, vol. 14: *Asbestos*, Lyon, France: 14–17 December 1976 (Lyon: IARC, 1977); and John M.G. Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature* (Edinburgh: Institute of Occupational Medicine, December 1980). Dr. Davis concluded from his review of the literature that "Animal experimental studies suggest that chrysotile is more fibrogenic and produces more bronchial carcinomas following inhalation than the amphibole varieties." (p. 17.) See also, John M.G. Davis, "The Use of Animal Experiments in the Study of Asbestos Bioeffects," *Hefte Unfallheilkd* 126 (November 1975): 564–574.

[158] Weill, Hughes, and Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," pp. 351–352.

Table 5.21

Mean Asbestosis Scores* of Sacrificed Rats

Length of Exposure	Amosite	Anthophyllite	Crocidolite	Chrysotile (Canadian)	Chrysotile (Rhodesian)	Control
8 weeks	2.0	2.0	2.0	2.0	2.7	1.3
3 months	2.5	2.7	2.8	2.7	3.0	1.3
6 months	2.2	3.2	2.6	3.0	2.6	1.2
12 months	4.0	5.2	4.3	4.3	4.3	–
18 months	–	–	–	–	–	1.2
24 months	4.3	6.2	4.8	6.0	5.8	1.8
6 months (after 18 months non-exposure)	3.2	5.0	3.7	5.5	3.7	–

Notes: *1: nil; 2: minimal; 4: slight; 6: moderate; 8: severe.

SOURCE: J. Christopher Wagner et al., "The Effects of the Inhalation of Asbestos in Rats," *British Journal of Cancer* 29:3 (March 1974): 262 (Table V).

Table 5.22

The Effects of Fibre Number on the Levels of Lung Damage Produced by the UICC Samples of Chrysotile, Amosite, and Crocidolite

Asbestos Type	Dust Cloud Mass (mg/m^3)	Fibre Number (ml^{-1})	Interstitial Fibrosis at 29 Months (%)	Lung Tumours	
				Benign	Malignant
Chrysotile	10	1,950	9.1	7	8
Crocidolite	10	860	1.4	1	0
Amosite	10	550	2.6	2	0
Chrysotile	2	390	3.8	6	2
Crocidolite	5	430	0.8	2	0

SOURCE: John M. G. Davis, "The Use of Animal Inhalation Experiments in the Study of Asbestos Bioeffects," *Staub-Reinhalt. Luft* 40:10 (October 1980): 454 (Table 1).

Weill reported that the pattern which emerged from comparing SMRs indicated that the crocidolite-exposed groups, and particularly the maintenance workers, had a higher risk of lung cancer than the solely chrysotile-exposed group. The results of Dr. Weill's comparison are summarized in Table 5.23. Even accepting the possible confounding effect of intermittent, high exposures for the maintenance workers, the influence of crocidolite appears to have been significant.[159] Similarly, Henderson and Enterline, in their factory retirees study, were able to compare the differences in lung cancer mortality between those in their cohort working in the asbestos-cement industry who were exposed both to crocidolite and chrysotile while manufacturing asbestos-cement pipe and those exposed only to chrysotile while manufacturing asbestos-cement shingles and sheets.[160] The authors reported that the men employed in these two segments of the industry had much in common: exposure to silica occurred in both cases; the levels of dust exposure were similar and occurred during the same processes; and often the two types of products were produced at a single location. In short, the main difference in the manufacture of these two types of products appeared to be the fibre types used. To repeat the data from Henderson and Enterline's most recent report, the 97 crocidolite-exposed asbestos-cement pipe workers had a mean exposure of 230 mppcf-yrs and a lung cancer SMR of 521.7; the 305 asbestos-cement shingle and sheet workers exposed only to chrysotile had a mean exposure of 255 mppcf-yrs and a lung cancer SMR of only 230.8.[161] With regard to amosite asbestos, the results were far less conclusive. For the 58 retirees from the single plant where only amosite was used, the SMR for lung cancer was high at 363.6, but so was the mean exposure of 330 mppcf-yrs; moreover, in those plants using amosite in combination with other types of asbestos, the risk of lung cancer was not much different than that found among the retirees exposed to chrysotile alone. (See Table 5.24.)

Some further indication of a different health hazard, at least as between chrysotile and crocidolite in terms of lung cancer mortality, comes from a comparison of the experience of the Quebec chrysotile miners and the Western Australian crocidolite miners. For example, the lung cancer SMR, 20 or more years from first employment in Quebec for all those employees who had worked one year or more, was 128;[162] the lung cancer SMR, 15 or more years from first employment in Western Australia for all

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[159] RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 37–38.

[160] Henderson and Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," pp. 122–124.

[161] Ibid., p. 121. It may also be observed that mortality rates for pneumoconiosis-pulmonary fibrosis (generally asbestosis) did not follow a similar pattern. Those workers exposed to chrysotile alone had a mortality rate per 100,000 of 396.3, whereas those workers exposed both to chrysotile and crocidolite had a corresponding mortality rate of 215.0.

[162] J. Corbett McDonald, "Asbestos-Related Disease: An Epidemiological Review," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 588–589.

Table 5.23

Standard Mortality Ratios for Respiratory Malignancy by Fibre Type

Type of Exposure	Total Fibre Exposure Within 20 Years of Initial Employment (mppcf-mos)*			
	≤20	20-200	>200	Total
No crocidolite exposure	12/21.4 56	10/13.0 77	8/4.4 182	30/38.8 77
Intermittent exposure to crocidolite in pipe plant	2/0.2 1,000**	0/0.7 0	5/1.4 357**	7/2.3 304**
Steady employment in pipe plant with crocidolite exposure	1/1.0 100	1/1.9 53	7/2.9 241**	9/5.8 155

Notes: *Million particles per cubic foot-months.
**Significant at $p<0.05$.

SOURCE: Hans Weill, Janet Hughes, and Carmel Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," *American Review of Respiratory Disease* 120:2 (August 1979): 352 (Table 8).

Table 5.24

Respiratory Cancer Mortality by Type of Asbestos, 1,075 Men Retiring 1941-1967 and Followed Through 1973

Type of Asbestos	Number of Men	Mean Exposure (mppcf-yrs)	Predicted SMR	Observed		95% Confidence Limits for Observed SMR
				Deaths	SMR	
Amosite only	58	330	317.1	4	363.6	99.1- 929.9
Chrysotile only	754	244	260.6	40	246.9	176.4- 336.4
Amosite and chrysotile	98	274	280.3	4	181.8	49.5- 465.0
Chrysotile and crocidolite	112	209	237.5	12	461.5	237.9- 805.4
Asbestos-cement pipe	83	219	244.1	11	578.9	289.4-1,035.6
Others	29	180	218.4	1	142.9	3.6- 793.9
Amosite, chrysotile, and crocidolite	53	277	282.3	3	250.0	51.5- 731.0
Asbestos-cement pipe	14	296	294.8	1	250.0	6.3-1,388.9
Others	39	270	277.7	2	250.0	30.3- 902.5

SOURCE: Vivian L. Henderson and Philip E. Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 122 (Table 4).

employees who had similarly worked one year or more, was nearly double at 246.[163] Such a comparison between different cohorts with different dust exposures is admittedly fraught with uncertainty, but it does provide some evidence of the relative health risks between fibre types at least in asbestos mining.

And yet, a similar comparison between textile operations suggests quite a different conclusion. As we have already observed, of the three United States asbestos plants studied by Dr. A.D. McDonald, two manufactured textiles: the Charleston plant (studied by Dr. Dement as well), using only chrysotile, and the Pennsylvania plant, using all three main fibre types. In both plants, the cohorts were constituted on the same basis; in both plants, the employees on average worked for similar lengths of time; their smoking habits did not appear to differ greatly, and if anything the average exposures in Pennsylvania were higher at 2.32 mppcf as compared to 1.80 mppcf in Charleston. Yet the SMR for lung cancer 20 or more years from first employment for Dr. A.D. McDonald's entire cohort in Charleston was 199.5, while in Pennsylvania it was only 105; and in the highest dust exposure category, the figure was 1,031.9 in Charleston and 416.1 in Pennsylvania.[164] Not only was the lung cancer risk higher in the plant using chrysotile only, it is fair to say that amongst all of the various cohort studies that have been reported, none has had a higher lung cancer mortality rate than the Charleston chrysotile textile plant. This in itself is strong evidence of the fact that there is no consistent association between fibre type and health effects insofar as lung cancer mortality is concerned.

Moreover, as with asbestosis, the results of the animal evidence in relation to lung cancer do not indicate that chrysotile is any less hazardous than the amphiboles. Indeed, in several inhalation studies, chrysotile has had a rather worse malignant lung tumour record than the amphiboles. Thus, in the large inhalation experiment published by Wagner et al. in 1974 in which rats had been treated with UICC standard reference samples, Rhodesian chrysotile produced by far the greatest number of lung tumours, followed in order by Canadian chrysotile, crocidolite, and amosite.[165] The results of this study as they relate to the production of lung tumours are summarized in Table 5.25. In the later inhalation experiment by Dr. Davis,

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[163] Hobbs et al., "The Incidence of Pneumoconiosis, Mesothelioma and Other Respiratory Cancer in Men Engaged in Mining and Milling Crocidolite in Western Australia," p. 622.

[164] A.D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Textile Plant," Tables 4 and 5, p. 364; and A.D. McDonald et al., "Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture," Tables 4 and 5, p. 371.

[165] Wagner et al., "The Effects of the Inhalation of Asbestos in Rats."

Table 5.25

Number of Animals with Lung Tumours or Mesotheliomata

Exposure	Number of Rats at Risk*	Number of Malignant Lung Tumours	Number of Mesotheliomas
Amosite	146	11	1
Anthophyllite	145	16	2
Crocidolite	141	16	4
Chrysotile (Canadian)	137	17	4
Chrysotile (Rhodesian)	144	30	0
Control	126	0	0

Note: *Rats which survived at least 300 days after start of exposure.

SOURCE: Adapted from: J. Christopher Wagner et al., "The Effects of the Inhalation of Asbestos in Rats," *British Journal of Cancer* 29:3 (March 1974): 264 (Table VII).

all of the malignant lung tumours developed in the chrysotile-treated animals; none were produced by either crocidolite or amosite dust clouds.[166] (See Table 5.22 in this chapter.)

C.4 The Evidence in Relation to Mesothelioma

However, it is neither the incidence of asbestosis nor of lung cancer which has prompted the concern about amphibole asbestos exposure. Rather, the principal indictment against crocidolite and amosite has been based upon the demonstrated incidence of mesothelioma among cohorts of asbestos workers exposed to these fibre types and the comparatively rare occurrence of the tumour in chrysotile-exposed populations.

Perhaps the most compelling evidence comes from the experience of a small group of gas mask workers employed during World War II in both Canada and England. The health experience of the Canadian workers has been studied by Dr. A.D. McDonald and Dr. J.C. McDonald, who reported on a cohort of 199 persons exposed to crocidolite between 1939 and 1942 in three separate factories.[167] In two of the plants, both located in Montreal, filter pads containing pure crocidolite were made, and in a third plant, located in Ottawa, these filters were packed into cannisters which were assembled into gas masks for use by the Canadian army. These cannisters contained crocidolite, 20% by weight, and wool, 80% by weight. Of the 199 employees, 174 were traced to the end of 1975, by which time 56 had died. Out of these 56 deaths, 9 were from mesothelioma (and, it may be noted, 7 from lung cancer). This statistic provides a stark contrast to the experience of the Quebec chrysotile miners. In the Quebec mines, a cohort of 11,379 workers, many of whom were long-term employees, yielded but 11 deaths from mesothelioma. Among the gas mask workers, a cohort approximately 1/60 in size, none of whom were employed longer than 3 years, produced almost the same number of mesotheliomas (and, it may be observed parenthetically, a lung cancer risk twice as high). There was also an important difference in the site of the tumours between the two cohorts: all 11 of the mesotheliomas among the chrysotile miners were pleural, whereas 6 of the 9 mesotheliomas among the crocidolite gas mask workers were peritoneal.[168] Table 5.26 compares malignant disease in the chrysotile miners and millers and in the crocidolite gas mask workers to the end of 1975.

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[166] Davis, "The Use of Animal Experiments in the Study of Asbestos Bioeffects," p. 454.

[167] Alison D. McDonald and J. Corbett McDonald, "Mesothelioma After Crocidolite Exposure During Gas Mask Manufacture," *Environmental Research* 17 (December 1978): 340-346.

[168] Ibid., pp. 343-344.

Table 5.26

Malignant Disease in Chrysotile Miners and Millers and in Crocidolite Gas Mask Filter Workers to the End of 1975

	Crocidolite Gas Mask Filter Workers	Chrysotile Minars and Millers
Total cohort	199 (55% male)	11,379 (96% male)
Traced	174	10,259
Dead	56	4,247 (since 1935)
Cancer of the lung or bronchus	7 (13%)	242 (6%)
Mesothelioma	9 (16%)*, ***	11 (0.26%)**, ***
Other malignant disease	7 (13%)	632 (14%)

Notes: *Four certified; two diagnosed at autopsy; three after pathologist's review.
**Eight certified, one of which was not thought by the pathologist to be a mesothelioma; three diagnosed at autopsy.
***Two cases were common to both series * and ** above.

SOURCE: Alison D. McDonald and J. Corbett McDonald, "Mesothelioma After Crocidolite Exposure During Gas Mask Manufacture," *Environmental Research* 17 (December 1978): 344 (Table 3).

The disease record of the Canadian gas mask workers was approximately matched by a group of female employees assembling gas masks at a factory in Nottingham, England, during the war.[169] In fact, there were two types of gas masks made at the English factory: military masks, which as in Canada used crocidolite filters and were assembled over a period of 4½ years; and civilian masks, which used chrysotile filters and were made on a limited scale for a period of 5 months. The mortality experience of these workers has been studied and reported on by Jones, Pooley, and Smith beginning in 1976.[170] Due to the small number of workers producing civilian masks and their limited asbestos exposure, the authors declined to draw any conclusion about their health experience. But the crocidolite-exposed women responsible for assembling military masks have been followed to the end of 1978. Of the cohort of 951 employees, only 578 have been traced. Among this group, there have been 166 deaths, of which 17 or fully 10% have died of mesothelioma. The authors reported that 13 of the mesotheliomas were pleural and only 4 peritoneal, a somewhat different proportion than found in the Canadian cohort.[171]

The incidence of mesothelioma among the crocidolite-exposed gas mask workers, compelling as it may be, does not stand alone. The Western Australian crocidolite miners have produced more than twice as many mesotheliomas as the Quebec chrysotile miners in a cohort little more than half the size.[172] The incidence of mesothelioma at and in the vicinity of the Cape crocidolite mines in South Africa, while not the subject of any systematic study, is on its face dramatically different from the experience of the asbestos miners and residents of the Eastern Townships in Quebec. In addition to the substantial number of mesotheliomas occurring among the Cape crocidolite miners themselves, Professor Webster, in the period 1956–1982, reported 178 cases of mesothelioma due to domestic or neighbourhood exposure.[173] By contrast, despite the extensive mining of chrysotile asbestos in Quebec and the extensive investigation of the incidence of mesothelioma in that province, there is a notably lower incidence of the disease among those living or working in the mining area. In her study of malignant mesothelioma in Quebec between 1960 and 1978, Dr. A.D. McDonald reported 6 cases among family members of Quebec miners and millers and 2 persons with the disease who had lived within 20 miles of the chrysotile mines.[174]

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[169] J.S.P. Jones et al., "The Consequences of Exposure to Asbestos Dust in a Wartime Gas-Mask Factory," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 637–653.

[170] J.S.P. Jones, F.D. Pooley, and P.G. Smith, "Factory Populations Exposed to Crocidolite Asbestos — A Continuing Survey," in *Environmental Pollution and Carcinogenic Risks*, eds. C. Rosenfeld and W. Davis, IARC Scientific Publications, no. 13 (Lyon, France: International Agency for Research on Cancer, 1976), pp. 117–120.

[171] Jones et al., "The Consequences of Exposure to Asbestos Dust in a Wartime Gas-Mask Factory," p. 645. Among these female workers, there was also an excess of lung cancer, with 12 observed as against 6.3 expected, but no excess of any other type of malignant disease, including gastrointestinal cancer.

[172] See Section B.1 of this chapter.

This evidence is admittedly circumstantial. But there is more direct evidence. At least 8 of the 10 mesotheliomas (all of which were pleural) at the Ferodo plant have been attributed by Berry and Newhouse to the crocidolite exposure in a plant using predominantly chrysotile and where otherwise there was little or no evidence of disease.[175] In A.D. McDonald's study of the three United States asbestos factories, among the over 2,000 deaths in the two factories using chrysotile, there has been only one mesothelioma, whereas among the 1,400 deaths in the factory using a combination of chrysotile and the amphiboles, there have been no less than 14 mesotheliomas (10 pleural and 4 peritoneal), with a distinct possibility of other unrecognized peritoneal tumours.[176] The amphibole-exposed naval shipyard workers in England studied by Rossiter and Coles produced 31 mesotheliomas (and 9 deaths from asbestosis) but otherwise little evidence of increased mortality among the 1,043 deaths in the total cohort, suggesting that while the exposure of these workers may not have been heavy, it was particularly likely to induce mesothelioma.[177] (See Table 5.27.) There were 21 mesotheliomas at the Scarborough asbestos-cement factory studied by Finkelstein (of which 9 were peritoneal), all but one of which had crocidolite exposure; whereas at the Cardiff asbestos-cement factory investigated by Thomas et al. at which only chrysotile was used, save for a two-year period in the 1930s when there was some crocidolite, there were only 2 mesotheliomas (both pleural) in a cohort approximately 3 times as large as that at Scarborough, and both of those likely had crocidolite exposure.[178]

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[173] Letter from Professor Ian Webster to the Royal Commission on Asbestos, 14 July 1983. See also, J.M. Talent et al., "A Survey of Black Mineworkers of the Cape Crocidolite Mines," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 723-729. It would appear that over 90% of the environmental cases reported by Professor Webster have been exposed to Cape crocidolite. Telephone communication between Professor Ian Webster and Royal Commission on Asbestos Staff, 27 October 1983. See also, J. Corbett McDonald, "Exposure Relationships and Malignant Mesothelioma," in *Proceedings of Asbestos Symposium*, Johannesburg, South Africa: 3-7 October 1977, ed. H.W. Glen (Randburg: National Institute for Metallurgy, 1978): 79. (Discussion.)

[174] A.D. McDonald, "Malignant Mesothelioma in Quebec," pp. 675-676. In the article, Dr. A.D. McDonald also compared the incidence of mesothelioma among the chrysotile miners and millers with the incidence of mesothelioma in four asbestos factories in the province of Quebec. Twenty-two cases of mesothelioma occurred within 50 years of employment in three of the factories, all of which used amphibole asbestos at various points in time and each of which apparently had an annual workforce of under 400. There were no cases of mesothelioma in the fourth factory (with an approximate annual employment of 140) which used only chrysotile and, at the time of the article, 16 cases of mesothelioma attributable to chrysotile exposure in the Quebec mines and mills, whose average annual employment was approximately 6,000.

[175] Berry and Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos."

[176] See Sections B.2 and B.3 of this chapter.

Among the cohort of 820 amosite insulation workers at Paterson, New Jersey, there have been (according to best evidence) 14 mesotheliomas, half of them peritoneal.[179] And, finally, of 2,271 deaths among the North American insulators exposed to chrysotile and amosite and studied by Dr. Selikoff, 175 died of mesothelioma (112 peritoneal and 63 pleural).[180] Again, to put this last statistic in perspective, the cohort of insulators studied by Dr. Selikoff had approximately one-half the number of deaths as occurred among the Quebec miners, but 16 times as many mesotheliomas.

The epidemiological evidence, at first blush overwhelming, should at least be qualified to some extent. In most instances, there is little or no direct evidence of the fibre concentrations to which the various cohorts we have discussed have been exposed. Accordingly, there is at least the possibility that what may appear to be the effect of fibre type may in whole or in part be the effect of high exposure levels. What appears to be a low incidence of mesothelioma in a particular cohort may reflect not the fibre type that was used, but the fact that exposure levels in the cohort were such

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[177] Charles E. Rossiter and Ruth M. Coles, "HM Dockyard, Devonport: 1947 Mortality Study," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 713-721. See also, J.C. McDonald, "Asbestos-Related Disease: An Epidemiological Review," p. 592; P.G. Harries, "Experience with Asbestos Disease and Its Control in Great Britain's Naval Dockyards," *Environmental Research* 11:2 (April 1976): 261-267; and Geoffrey Sheers and Ruth M. Coles, "Mesothelioma Risks in a Naval Dockyard," *Archives of Environmental Health* 35:5 (September/October 1980): 276-282. Crocidolite asbestos was widely applied by a spray process for environmental insulation and fire protection between approximately 1940 and the early 1960s. Between 1950 and 1963, there was a large increase in the amount of amosite used for machinery insulation. Harries noted that there had been 55 deaths from mesothelioma between 1964 and 1973 at the Royal Navy Dockyard at Plymouth, of which all but one were pleural. Sheers and Coles reported 108 cases of mesothelioma at this dockyard between 1964 and mid-1978, of which all but 3 were pleural.

[178] See Section B.4 of this chapter.

[179] See Section B.5 of this chapter. Even so, the ratio of mesothelioma cases to lung cancer cases is quite low in this cohort. Dr. A.D. McDonald in her testimony suggested this was likely because the Paterson, New Jersey, workers were, as a group, much older than is usual. RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 59-60.

[180] See Section B.6 of this chapter.

Table 5.27

Observed and Expected Deaths, by Cause, Among Dockyard Workers

Cause of Death	Number of Deaths	England and Wales		South-west Estimates**		
		Expected	SMR*	Expected	SMR	
All deaths	1,043	1,081.2	96	998.4	104	
All cancers	265	282.1	94	255.6	104	
Mesothelioma	31***	0.5	6,400	0.4	7,700	$P \ll 0.001$
Lung cancer	84***	119.7	70	100.3	84	
Gastrointestinal cancer	63	83.3	76	76.2	83	
Others	87†	78.6	111	78.7	111	
Asbestosis, pulmonary fibrosis	9	0.03	27,000	0.03	32,000	$P \ll 0.001$
Other respiratory diseases	67	97.3	69	81.9	82	
Circulatory diseases	518†	464.2	112	440.8	118	$P < 0.001$
Other causes	174	237.6	73	220.1	79	$P < 0.01$
Unknown causes	10	–	–	–	–	

Notes: *SMR means standardized mortality ratio.

**Estimated death rates in south-west Britain (including Devonport).

***Three also had asbestosis.

†One also had asbestosis.

SOURCE: Charles E. Rossiter and Ruth M. Coles, "HM Dockyard, Devonport: 1947 Mortality Study," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), Table 2, p. 717.

that, overall, there was not much disease. There were clearly few mesotheliomas among the Quebec miners given the size of the cohort, but the overall excess of lung cancer was also relatively low. Mr. Peto has in fact demonstrated that the ratio of excess lung cancer to mesothelioma (at least to pleural mesothelioma) is quite similar among most studies irrespective of what fibre type or types have been utilized.[181] (See Table 5.28.) Still, this ratio does not hold either for the English shipyard workers studied by Rossiter and Coles or for the Charleston textile workers.[182]

Further, among factory workers exposed to mixed fibre types, it may be inappropriate to attribute the incidence of mesothelioma to amphibole exposure when in fact chrysotile is invariably the predominant fibre used.[183] Mr. Peto has argued that the crocidolite exposure at Rochdale was minimal, yet the incidence of pleural mesothelioma was similar to that of the East London asbestos plant studied by Newhouse and Berry where crocidolite exposure was heavy, thus suggesting it is unlikely that crocidolite alone was responsible for the mesothelial tumours at Rochdale.[184] Further, the lack of

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[181] RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 61–63. See also, J. Corbett McDonald and Alison D. McDonald, "Mesothelioma as an Index of Asbestos Impact," in *Banbury Report 9: Quantification of Occupational Cancer*, eds. Richard Peto and Marvin Schneiderman ([Cold Spring Harbor, New York]: Cold Spring Harbor Laboratory, 1981), Table 2, pp. 76–77.

[182] In the Rossiter and Coles study, there were 31 cases of mesothelioma (likely all pleural) and no excess lung cancer. Dr. Dement had no cases of pleural mesothelioma, one peritoneal mesothelioma, and 18.5 excess lung cancer cases. Mr. Peto himself indicated in his testimony that the Rossiter and Coles study was difficult to explain. RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 61–63.

[183] Julian Peto, Herbert Seidman, and Irving J. Selikoff, "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment," *British Journal of Cancer* 45 (1982): 124–135. The authors indicated that amphiboles are largely responsible for asbestos-related peritoneal mesothelioma, but went on to state:

> It may therefore be dangerously optimistic to attribute the substantial incidence of pleural mesothelioma among chrysotile factory workers to occasional crocidolite exposure, merely because mesothelioma is rare among chrysotile miners. . . . The overall excess of lung cancer is also relatively low among chrysotile miners, and the only safe conclusion must be that dose-response relationships cannot be expected to apply outside the environment in which they are established, at least until the range of fibre sizes to be included in the fibre count has been chosen less arbitrarily. (pp. 134–135.)

[184] Peto, "The Hygiene Standard for Chrysotile Asbestos," p. 488. See also, Muriel L. Newhouse and Geoffrey Berry, "Patterns of Mortality in Asbestos Factory Workers in London," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 53–60. This East London asbestos factory, studied by Newhouse and Berry, opened in 1913, at first producing chiefly asbestos textiles but later producing insulation materials and a variety of other products. Crocidolite asbestos was used until the late 1950s and chrysotile and amosite were also used. The factory closed in 1968. In the latest mortality study, the authors reported 775 deaths among the male workers, a cohort of 4,600. An analysis of the 545 deaths that occurred among the factory workers, excluding laggers, revealed 103 deaths from lung cancer, an excess of 60 over expected; and 46 deaths from mesothelioma, of which 19 were pleural.

Table 5.28

Ratio of Excess Lung Cancer to Mesothelioma

Fibre	Lung and Pleural Cancer		(A)	Mesothelioma (B)	Mesothelioma/Excess (B)/(A)
	O	E	O-E	O	
Chrysotile and Crocidolite (Enterline)	14	5.2	8.8	3	34%
	49	18.1	30.9	2	6%
Insulation (Selikoff)	533	105.6	427.4	175	41%
Crocidolite Mine (Hobbs)	56	28.7	27.3	9	33%
Chrysotile and Crocidolite(?) (Peto)	47	23.3	23.7	7	30%
Chrysotile Mine (J.C. McDonald)	230	184.0	46.0	11	24%
Mixed (Newhouse)	180	48.8	131.2	56	43%
Amosite (Seidman)	116	19.1	96.9	16	17%

SOURCE: Ontario, Royal Commission on Asbestos, Exhibit II-37, Tab 12, in RCA Transcript of Public Hearings, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 10, 61-63: Julian Peto, Assorted Transparencies. (Mimeographed.)

reported mesotheliomas among the Transvaal amosite and crocidolite miners provides some evidence that the amphiboles do not always produce a high incidence of this tumour.

And yet, even with these qualifications, there are three inescapable conclusions that in our judgement emerge from the epidemiological evidence. First, while chrysotile is capable of causing mesothelioma in humans, the incidence of mesothelioma among chrysotile-exposed cohorts has been relatively low. No better example may be found than the Charleston textile plant with an extraordinarily high incidence of lung cancer and yet but one mesothelioma. Second, amphibole exposure is at least capable of causing a significant incidence of mesothelioma in humans. The experiences of the gas mask workers, the North American insulators, and the English shipyard workers by themselves are eloquent testimony to this proposition. Third, the amphiboles appear to be almost solely responsible for the incidence of peritoneal (as opposed to pleural) mesothelioma among asbestos workers.[185] Almost without exception, the mesotheliomas that have been diagnosed in cohorts exposed only to chrysotile have been pleural.[186] Exposure to the amphiboles has not produced any consistent pattern. On the one hand, the mesotheliomas in the Western Australian crocidolite mines and at the English dockyards were nearly all pleural. On the other hand, two-thirds of the tumours among the Canadian gas mask workers and the North American insulators were peritoneal.[187] On balance, however, the observed incidence of tumours of the peritoneum has been associated with amphibole exposure. The possibility remains that this site is particularly susceptible to amosite exposure as evidenced by the experience of the amosite factory workers at Paterson, New Jersey, and the North American insulators.

The epidemiological evidence from cohort studies and case-series reports of the effect of fibre type on the incidence of mesothelioma is supported by the lung tissue studies that have been reported in the literature. For example, Jones et al. reported on a study of 93 cases of persons dying from mesothelioma in England in 1976.[188] At autopsy, lung tissue was available for analysis from 86 of these cases and also from 56 matched controls. The authors found that amphibole fibres, both crocidolite and amosite, occurred more frequently and in greater amounts in the lungs of the mesothelioma cases than the controls. Chrysotile fibres, on the other hand, were not present in any greater number of the cases than in the controls, and indeed in 30 of the cases of mesothelioma there were no chrysotile fibres at all.[189] Similar findings have been reported by A.D. McDonald, J.C. McDonald, and Pooley in their study of the mineral fibre content of lung tissue from North American mesothelioma victims in 1972.[190] They examined 99 cases of mesothelioma and matched controls. As in the English study, roughly equal quantities of chrysotile fibres were found in the cases and the controls; by contrast, higher quantities of amosite were found in the cases than in the controls, and higher quantities of crocidolite were likewise found in the cases than in the controls. The results of this study are set out in Table 5.29. These results, as the authors have fairly pointed out, must be treated with some caution by reason of the likelihood that chrysotile fibres disappear over the course of time.[191] Still, if chrysotile were responsible for a significant proportion of the mesothelioma tumours in this series, one would expect higher counts of this fibre in the lung tissue of the cases than were found.

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[185] See Peto, Seidman, and Selikoff, "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment," pp. 130-131, where the authors reached a similar conclusion and suggested that the rigidity of the amphiboles may be a necessary prerequisite for migration to the peritoneum.

[186] Curiously, the one mesothelioma at Charleston, South Carolina, was peritoneal.

[187] See Peto, Seidman, and Selikoff, "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment," p. 134, where the authors suggested that these different ratios likely reflect differences in fibre dimension rather than chemical structure.

[188] J.S.P. Jones et al., "The Pathology and Mineral Content of Lungs in Cases of Mesothelioma in the United Kingdom in 1976," in *Biological Effects of Mineral Fibres*, vol. 1, pp. 187-199.

Thus, the cumulative effect of all of the epidemiological evidence — cohort studies, case-series reports, and case-control studies of fibre content of lung tissue — presents a demonstrably convincing case against the amphiboles in relation to the incidence of mesothelioma. However, the animal evidence appears to present a much different picture. And it is the results of experimental studies with animals that have played an important role for those experts and regulatory agencies that do not believe there are substantial differences between the health hazards posed by chrysotile exposure, on the one hand, and amphibole exposure, on the other.[192] Again, it is not simply the fact that all fibre types have been able to induce mesothelioma in rats, whether the animals were dosed by inhalation or injection. In virtually all of the animal studies, chrysotile has produced at least as many or more mesothelial tumours than either crocidolite or amosite. The studies that have been carried out are far too numerous to permit us to report on all of them in detail. We choose to cite here the findings of a few of the experiments which we believe to be representative of the results of the vast majority of the studies.

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[189] Ibid., p. 198.

[190] Alison D. McDonald, J. Corbett McDonald, and Fred D. Pooley, "Mineral Fibre Content of Lung in Mesothelial Tumours in North America," *Annals of Occupational Hygiene* 26:1–4 (1982): 417–422.

[191] RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 35-36; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 100.

[192] See, for example, RCA Transcript, Evidence of Mr. Richard A. Lemen, 8 July 1981, Volume no. 17, pp. 122–124.

Table 5.29

Number (in Millions) of Asbestos Fibres per Gram of Dried Lung Tissue in 76 Male and 23 Female Cases of Mesothelioma in North America and Matched Controls

Number of Fibres	Chrysotile		Amosite		Crocidolite		Anthophyllite		Tremolite	
	Cases	Controls	Cases	Controls	Cases	Controls	Cases	Controls	Cases	Controls
Males										
None	3	1	37	47	47	60	58	61	29	31
<1	8	11	13	21	14	11	11	12	33	28
1<10	38	38	14	7	6	5	4	3	10	16
10<100	25	22	8	1	8	—	2	—	3	1
100<1,000	2	2	3	—	1	—	—	—	—	—
1,000 and over	—	2	1	—	—	—	—	—	1	—
Females										
None	—	—	17	15	15	16	18	17	7	9
<1	—	5	4	7	6	6	2	5	9	8
1<10	12	11	2	1	2	1	3	1	7	6
10<100	10	7	—	—	—	—	—	—	—	—
100 and over	1	—	—	—	—	—	—	—	—	—

SOURCE: Alison D. McDonald, J. Corbett McDonald, and Fred D. Pooley, "Mineral Fibre Content of Lung in Mesothelial Tumours in North America," *Annals of Occupational Hygiene* 26:1-4 (1982): 418 (Table 1).

In an early intrapleural injection experiment reported by Wagner and Berry in 1969, mesothelioma developed in 61 of the 96 rats given chrysotile, 55 of the 94 given crocidolite, and in only 38 of the 96 given amosite.[193] In an experiment reported by Reeves et al. in 1971, no mesotheliomas were produced by inhalation. But when the rats were injected with different types of asbestos, chrysotile produced 5 mesotheliomas; crocidolite, 4; and amosite, none.[194] A large 1974 inhalation experiment by Wagner et al. resulted in one mesothelioma (among 146 rats) from amosite and 4 mesotheliomas each from crocidolite (141 rats) and Canadian chrysotile (137 rats), although none from Rhodesian crocidolite (144 rats).[195] (See Table 5.25 in this chapter.) And in an often-cited intrapleural injection study, reported on by Wagner, Berry, and Skidmore in 1974, mesothelioma developed in 66% of the rats implanted with a superfine Canadian chrysotile, 61% implanted with UICC crocidolite, 36% implanted with UICC amosite, 30% implanted with UICC Canadian chrysotile, and 19% implanted with Rhodesian chrysotile.[196]

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[193] J. Christopher Wagner and Geoffrey Berry, "Mesotheliomas in Rats Following Inoculation with Asbestos," *British Journal of Cancer* 23 (1969): 567–581.

[194] See Andrew L. Reeves et al., "Experimental Asbestos Carcinogenesis," *Environmental Research* 4 (1971): 496–511. The mortality and malignancy incidence of the rats exposed to asbestos in the experiment was as follows:

		Amosite					Crocidolite					Chrysotile				
		Inventory					Inventory					Inventory				
		0	6	12	24	Incidence of Malignancy	0	6	12	24	Incidence of Malignancy	0	6	12	24	Incidence of Malignancy
Species	Route	Months					Months					Months				
Rats	Inhalation	77	67	63	42	—	69	61	61	31	2 pulmonary cancers* both at 29 months	60	49	49	40	—
	Intratracheal	16	10	6	1	—	13	6	3	1	–	15	8	6	0	—
	Intrapleural	15	6	4	0	–	13	5	2	0	1 pleural mesothelioma** at 16 months	12	6	4	1	2 pleural mesotheliomas** at 9 and 14 months
	Intraperitoneal	11	3	0	0	–	13	7	4	0	3 peritoneal mesotheliomas*** at 12, 13, and 17 months	13	7	0	0	3 peritoneal mesotheliomas*** at 7, 9, and 11 months

Notes: *Pulmonary carcinoma (squamous cell pattern)
**Pleural mesothelioma (fibrosarcoma pattern)
***Peritoneal mesothelioma (fibrous, papillary, osteogenic, or epithelial patterns)

SOURCE: Andrew L. Reeves et al., "Experimental Asbestos Carcinogenesis," *Environmental Research* 4 (1971): 502 (Table 4, excerpt)

See also, Andrew L. Reeves, Henry E. Puro, and Ralph G. Smith, "Inhalation Carcinogenesis from Various Forms of Asbestos," *Environmental Research* 8 (1974): 178–202.

[195] Wagner et al., "The Effects of the Inhalation of Asbestos in Rats."

[196] J. Christopher Wagner, Geoffrey Berry, and Joseph W. Skidmore, "Studies of the Carcinogenic Effects of Fiber Glass of Different Diameters Following Intrapleural Inoculation in Experimental Animals," in *Occupational Exposure to Fibrous Glass: A Symposium*, College Park, Maryland: 26–27 June 1974, HEW Publication no. (NIOSH) 76–151 (Washington, D.C.: U.S. Department of Health, Education and Welfare, April 1976), Table 18-3, p. 196. See also, World Health Organization, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, vol. 14: *Asbestos*, Table 17, p. 47.

The animal evidence, therefore, in striking contrast to the epidemiological evidence, appears to afford no basis for suggesting that chrysotile is any less hazardous in relation to the production of mesothelioma than the amphiboles. And the thrust of this animal evidence is consistent with the various *in vitro* or cell culture studies that have been reported, most of which indicate that chrysotile is at least as cytotoxic and haemolytic as the amphiboles.[197] In short, the human evidence and the experimental evidence appear to be quite inconsistent. We shall address this inconsistency later in this chapter.

C.5 The Evidence in Relation to Gastrointestinal Cancer

As we have already observed, the association between asbestos exposure and cancer of the gastrointestinal tract appears to be less consistent and less marked when it has occurred among various cohorts of asbestos workers that have been studied than the association between asbestos and lung cancer, mesothelioma, or asbestosis.[198] However, when the incidence of gastrointestinal cancer is viewed in terms of fibre type, a somewhat clearer pattern emerges, although the type of exposure does not appear to us to explain completely the observed results. Our review of the available evidence does suggest that the finding of a dose-response relationship for gastrointestinal cancer and the existence of elevated mortality rates most frequently occur among amphibole-exposed populations. The incidence among the amosite factory workers at Paterson, the North American insulators, and the Belfast shipyard workers is demonstrably high;[199] and there have been excess deaths from abdominal cancers among a number of other industrial cohorts — the Scarborough cement workers, the Pennsylvania textile workers, the Manville retirees, and the East London factory workers[200] — exposed to mixtures of chrysotile and amphiboles. Still, there was no excess among the gas mask workers[201] or among the shipyard workers studied by Rossiter and Coles. Moreover, solely chrysotile-exposed populations have produced an excess of tumours from sites in the gastrointestinal tract. The SMR for abdominal cancer at Charleston was slightly elevated in Dr. Dement's cohort and even more so in Dr. A.D. McDonald's larger cohort; and even among the Quebec miners there was a substantial excess of cancer of the upper gastrointestinal tract among the most heavily exposed employees (although there was no excess at all in the cohort of cancer of the lower gastrointestinal tract).

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[197] Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, p. 17.

[198] See Chapter 2, Section D.4 of this Report.

[199] J.C. McDonald, "Asbestos-Related Disease: An Epidemiological Review," p. 594.

[200] In their latest report on the Manville retirees, Henderson and Enterline reported an SMR for gastrointestinal cancer of 137.8. Among the East London factory workers, there was a statistically significant excess of gastrointestinal cancer only among those workers most heavily exposed. (And, it may be noted, the authors included the peritoneal mesotheliomas with the gastrointestinal tumours.) For actual data on the relative risk of gastrointestinal cancer and the various cohorts discussed in this section, see Section B of this chapter.

[201] Jones et al., "The Consequences of Exposure to Asbestos in a Wartime Gas-Mask Factory," pp. 644-645. Among the women workers in the English gas mask factory, there were 10 cases of gastrointestinal cancer as against 20.3 expected.

On the other hand, there was no excess of gastrointestinal tumours at the Ferodo plant, none at the Cardiff asbestos-cement factory, and none among the Italian miners. There was no consistent dose-response relationship at the Connecticut friction products plant, and no excess of gastrointestinal tumours at Rochdale where, at least according to Mr. Peto, crocidolite exposure was minimal.[202] The cumulative effect of this evidence at least suggests, albeit not strongly, that an excess of gastrointestinal cancer is more likely to occur where there is amphibole exposure either alone or in conjunction with chrysotile than it is with chrysotile alone. This suggestion is consistent with the observed fact that there is a virtual absence of abdominal or peritoneal mesotheliomas among workers exposed only to chrysotile.

C.6 The Evidence in Relation to Crocidolite and Amosite

Thus far we have focused our discussion on the differences, if any, between the health hazards of chrysotile exposure, on the one hand, and amphibole exposure, on the other. We have not discussed whether there is any evidence which might suggest that the health hazards of crocidolite and amosite are different. It is to this subject that we now turn.

It is generally thought that as between the two fibre types, crocidolite poses a greater danger to health than amosite, and this view is indeed reflected in the present Ontario Regulation Respecting Asbestos. There is a considerable amount of evidence that argues in support of that view. There appears to be a very high incidence of mesothelioma in crocidolite mining and a very low incidence in amosite mining; the considerable number of mesotheliomas observed among various cohorts of shipyard workers has been largely attributed to crocidolite exposure;[203] the crocidolite-exposed gas mask workers and Dr. Finkelstein's cohort of Scarborough cement workers had a remarkably high disease incidence; and in animal experiments, crocidolite-exposed (and, for that matter, chrysotile-exposed) rats have invariably had a far worse record of tumour production than rats exposed to amosite.

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[202] J.C. McDonald, "Asbestos-Related Disease: An Epidemiological Review," p. 594. See also, note 57, *supra*.

[203] J.C. McDonald, "Asbestos-Related Disease: An Epidemiological Review," p. 592. See also, Harries, "Experience with Asbestos Disease and Its Control in Great Britain's Naval Dockyards," p. 261. Amosite asbestos was also used in ship construction and may also be implicated in the relatively high incidence of mesothelioma among various shipyard workers. This may be particularly true in the United States where arguably amosite was more widely used than crocidolite in shipyards.

Weighed against this evidence is the health experience of the North American insulators and the makers of insulation products. Admittedly the cohort at Paterson, New Jersey, had certain unusual features associated with it, but the fact remains that these amosite insulation workers had a particularly bad disease record, as we have previously discussed. The large cohort of North American insulators followed by Dr. Selikoff similarly had a high incidence of asbestos-related disease. The substantial number of mesotheliomas at the Pennsylvania textile factory arguably was attributable largely to the extensive use of amosite at that plant to make insulation products for the United States Navy. That part of the Henderson and Enterline cohort of retirees exposed solely to amosite, while admittedly small in number, had an excess incidence of lung cancer which closely approximated that part of the cohort exposed to crocidolite.[204] And lung tissue studies appear to support the proposition that amosite has played a significant role in the incidence of mesothelioma in North America.[205]

What is clear is that both amosite and crocidolite have resulted in serious disease records where utilized. If it is difficult to draw any definitive conclusions about the relative health hazards of amosite and crocidolite, this is because there are fewer studies of amosite-exposed populations than of crocidolite-exposed populations and also because there is an absence of actual exposure information in two principal cohorts where amosite has been used (the Paterson plant and the North American insulators). However, with respect to the latter deficiency, it is possible to derive exposure estimates, and we rely on such derived estimates in our risk assessment in Chapter 7. This assessment sustains the possibility that crocidolite is somewhat more hazardous than amosite. That possibility is further strengthened by evidence addressed in Section D of the present chapter. On balance, we conclude that the available evidence indeed suggests that crocidolite is somewhat more hazardous than amosite.

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[204] Henderson and Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," p. 122. Of the 1,075 retirees in the cohort, 58 were exposed to amosite only. There were 4 deaths from lung cancer and an SMR of 363.6.

[205] Alison D. McDonald, "Mineral Fibre Content of Lung in Mesothelial Tumours: Preliminary Report," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 681–685. See also, Table 5.29 in this chapter.

C.7 The Evidence in Relation to Anthophyllite, Tremolite, and Actinolite

The health evidence on these three fibre types is scanty. There is one cohort study of which we are aware on the health risks of anthophyllite: that of Meurman, Kiviluoto, and Hakama, who investigated the mortality experience of the Finnish anthophyllite miners.[206] They reported an overall lung cancer SMR of 167, rising to 333 in those with more than 10 years' exposure, but no cases of mesothelioma and no excess cancer of the digestive system. Neither actinolite nor tremolite has been used in sufficient quantities commercially to engage detailed investigation of their health effects. However, as we have already pointed out, tremolite is known to be present in certain deposits of chrysotile asbestos and talc.[207] Rowlands, Gibbs, and A.D. McDonald have recently reported on their investigation of the mineral fibre content of 47 lung samples from the Quebec miners and millers (none, however, being a victim of either asbestosis or mesothelioma). Their striking finding was that tremolite was found in approximately similar quantities to chrysotile in these lung samples.[208] Large quantities of tremolite were also found in a series of 20 lung samples previously examined by Pooley from the same cohort.[209] And Dr. J.C. McDonald, during his testimony, went so far as to speculate that the mesothelioma among the Quebec miners heretofore attributed to chrysotile may be due to tremolite exposure.[210] Kleinfeld, Messite, and Zaki have investigated the mortality experience of a group of 250 miners of a talc deposit contaminated by tremolite, and to a lesser extent by anthophyllite, located in upper New York State.[211] The authors reported increased lung cancer mortality and one death from peritoneal mesothelioma. While this evidence indicates a possible health risk from tremolite exposure, in our judgement there are not yet sufficient data to come to any firm conclusion. However, we do point out that to the extent that tremolite contaminates mines in Ontario, and to the extent that it is utilized commercially, its health effects warrant further scientific investigation.

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[206] L.O. Meurman, R. Kiviluoto, and M. Hakama, "Mortality and Morbidity Among the Working Population of Anthophyllite Asbestos Miners in Finland," *British Journal of Industrial Medicine* 31:2 (April 1974): 105-112. The authors' study cohort consisted of 1,092 miners first employed between 1936 and 1969, of whom 95% were traced and 248 had died.

[207] See Chapter 2, Section B.4 of this Report.

[208] Neil Rowlands, Graham W. Gibbs, and Alison D. McDonald, "Asbestos Fibres in the Lungs of Chrysotile Miners and Millers — A Preliminary Report," *Annals of Occupational Hygiene* 26:1-4 (1982): 411-417.

[209] Fred D. Pooley, "An Examination of the Fibrous Mineral Content of Asbestos Lung Tissue from the Canadian Chrysotile Mining Industry," *Environmental Research* 12 (1976): 281-298.

[210] RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 97-98. Dr. Davis testified that he had just completed an inhalation study with pure tremolite. While the study had been completed at the time of his testimony in January 1982, not all of the data had been analyzed. However, Dr. Davis suggested that his experiment appeared to indicate that tremolite is very harmful to rats and may be more harmful than chrysotile. See RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 96-97. Mr. Ross Hunt testified that tremolite may be every bit as much a concern as crocidolite and noted that in 3 cases of mesothelioma in Cyprus, tremolite was the only asbestos fibre found by Dr. Fred D. Pooley at autopsy. See RCA Transcript, Evidence of Mr. Ross Hunt, [illegible] June 1982, Volume no. 41, p. 31.

C.8 Summary

In the first section of this chapter we observe that the incidence of asbestos-related disease appears to vary among industrial processes. In this section we observe that, at least in respect of the incidence of mesothelioma, the epidemiological evidence indicates there is a marked difference between chrysotile and amphibole exposure. It is, we believe, also fair to suggest that while chrysotile alone is capable of producing a high incidence of disease, as at Charleston, there are some processes in which it appears to have been used with little health risk — principally friction materials and to a lesser extent mining. On the other hand, there is no cohort systematically studied of which we are aware which has been exposed to amphiboles alone or to amphiboles in combination with chrysotile that has not demonstrated a considerable excess mortality associated with asbestos exposure. And yet, we also observe that the experimental evidence produces a conclusion that is quite contrary to the epidemiological evidence. Not only do all fibre types produce all asbestos-related diseases in animals, no matter what type of experiment has been undertaken, but also chrysotile appears to be no less hazardous than amosite or crocidolite, and this includes the production of mesothelioma as well as lung cancer and fibrosis.

How then do we account for these differences? Why is it that some industrial processes appear to be less hazardous than others? Why do the amphiboles appear to be largely responsible for the incidence of mesothelioma in humans? Why do the experimental evidence and the epidemiological evidence differ so strikingly in relation to the incidence of mesothelioma? It is to these questions that we now turn.

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[211] Morris Kleinfeld, Jacqueline Messite, and Mahfouz H. Zaki, "Mortality Experiences Among Talc Workers: A Follow-up Study," *Journal of Occupational Medicine* 16 (1974): 345-349. See also, John M. Dement and Ralph D. Zumwalde, "Occupational Exposures to Talcs Containing Asbestiform Minerals," in *Dusts and Disease*, pp. 287-305.

D. Reconciling the Evidence — Fibre Dimension and Other Considerations

The questions which we pose appear to beg a further question: What characteristics of asbestos fibres determine their pathogenicity? In our judgement, the starting point is the recognition of the significance of the dimensions of the fibre — its length, its diameter, its shape — in causing disease. In Chapter 4 we have already indicated that the diameter of the fibre plays an important role in its ability to become airborne, and therefore its ability to be inhaled. The thinner the diameter of fibres, the more likely they are to create respirable dust clouds. The diameter of the fibre is also important in terms of its ability to enter the respiratory tract and to penetrate into the lung, the pleural and even the abdominal cavities. Here too, the thinner the fibre, the more easily it can penetrate. Shape affects the behaviour of fibres because fibres that are curly and not straight behave as if they are thicker than their actual fibre diameter would suggest. Length is important in terms of the respirability of the fibre; but even more significantly, once the fibre has been inhaled, it is important in terms of one of the body's principal defence mechanisms: the activity of the macrophages. Longer fibres, that is, fibres with a minimum length of at least 5 or perhaps 8 microns, are not readily engulfed and immobilized by macrophages, and this may be a factor in their causing disease. To put the point differently, there is a preferential clearance of shorter fibres from the body.

These considerations taken together strongly suggest that it is the long, thin asbestos fibres that are apt to be most pathogenic. The late Dr. Mearl F. Stanton and his colleagues at the National Cancer Institute in the United States came to exactly this conclusion in a pathbreaking series of animal experiments which investigated the relationship between fibre dimension and carcinogenicity.[212] Dr. Stanton and his colleagues implanted various durable fibres of different but respirable size on the pleura of rats. They found that the biologic activity of these fibres in relation to the incidence of mesothelioma was very much dependent upon their dimension. In the most recent of their studies, reported in 1981, their results indicated that the probability of mesothelioma correlated best with fibres 0.25 microns or less in diameter and 8 microns or more in length, but that a relatively high correlation was obtained for fibres of a dimension up to 1.5 microns in diameter and greater than 4 microns in length.[213] (See Table 5.30.) Experimental work by Dr. Friedrich Pott has also demonstrated that it is the long, thin fibres which are most pathogenic.[214] Thus, physics, biology, and experimental work all point to the significance of fibre dimension in causing asbestos-related disease, both in terms of the respirability of fibres and, once inhaled, in terms of their ability to reach the target site and to cause disease.

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[212] Mearl F. Stanton and Constance Wrench, "Mechanisms of Mesothelioma Induction with Asbestos and Fibrous Glass," *Journal of the National Cancer Institute (JNCI)* 48:3 (March 1972): 797–821; Mearl F. Stanton et al., "Carcinogenicity of Fibrous Glass: Pleural Response in the Rat in Relation to Fiber Dimension," *Journal of the National Cancer Institute* 58 (1977): 587–603; Mearl F. Stanton and Maxwell Layard, "The Carcinogenicity of Fibrous Minerals," in *Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods*, Gaithersburg, Maryland: 18–20 July 1977, NBS Special Publication 506 (Washington, D.C.: U.S. National Bureau of Standards, November 1978), pp. 143–150; and Mearl F. Stanton et al., "Relation of Particle Dimension to Carcinogenicity in Amphibole Asbestoses and Other Fibrous Minerals," *Journal of the National Cancer Institute* 67:5 (November 1981): 965–975.

And when consideration is given to fibre dimension in assessing the epidemiological and animal evidence, a more consistent and coherent picture begins to emerge. The amphiboles generally, and crocidolite in particular with its straight, thin, needle-like dimensions, have a greater ability to become airborne than chrysotile and therefore a greater ability to create a respirable dust cloud than chrysotile.[215] To use a common phrase, the amphiboles, and especially crocidolite, are dustier than chrysotile. As between crocidolite and chrysotile, experiments have demonstrated that chrysotile requires much more active disturbance to generate a dust cloud than does

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[213] Stanton et al., "Relation of Particle Dimension to Carcinogenicity in Amphibole Asbestoses and Other Fibrous Minerals," pp. 965, 973. In their earlier work, Dr. Stanton and his colleagues concluded that the most carcinogenic fibres were those longer than 8 microns and thinner in diameter than 1.5 microns. See RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, p. 43. Dr. Davis testified that the notion that a long fibre might be more hazardous than a short fibre goes back as far as the work of Professor E.J. King in 1946. See E.J. King, J.W. Clegg, and V.M. Rae, "Effect of Asbestos, and of Asbestos and Aluminium, on the Lungs of Rabbits," *Thorax* 1 (September 1946): 188-197.

[214] Friedrich Pott, "Some Aspects on the Dosimetry of the Carcinogenic Potency of Asbestos and Other Fibrous Dusts," *Staub-Reinhault. Luft* 38:12 (December 1978): 486–490. According to Dr. Eric J. Chatfield, while Dr. Pott suggested a declining carcinogenicity factor below 5 microns in length, he still indicated that fibres greater than 1 micron in length would have an effect. Dr. Chatfield observed that if Dr. Pott is correct, the overall effect of short fibres may be larger than the effect of longer fibres simply because there are more of them in any dust cloud. See RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, pp. 14–15. See also, Ontario, Royal Commission on Asbestos, Exhibit II-27 [hereafter RCA Exhibit], Tab 19, in RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18: Eric J. Chatfield, "Airborne Asbestos Fibres: A Summary of Some Measurement Problems," Sheridan Park, Ontario Research Foundation, 1981, p. 1. (Mimeographed.)

[215] See RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, p. 96; RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, p. 28; RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), p. 94; and RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 11.

Table 5.30

Correlation Coefficients of Logit of Tumour Probability with Common Logarithm of Number of Particles per Microgram in Different Dimensional Ranges

Fibre Diameter (Microns)	Fibre Length (Microns)		
	<4	>4-8	>8
>4	–	-0.28	-0.30
>1.5-4	-0.45	-0.24	0.13
>0.25-1.5	0.01	0.45	0.68
<0.25	0.20	0.63	0.80

SOURCE: Mearl F. Stanton et al., "Relation of Particle Dimension to Carcinogenicity in Amphibole Asbestoses and Other Fibrous Minerals," *Journal of the National Cancer Institute (JNCI)* 67:5 (November 1981): 973 (Table 3).

crocidolite.[216] Further, these experiments have also indicated that crocidolite will repeatedly become airborne, whereas when chrysotile is allowed to settle out on any horizontal or vertical surface, it is much less likely to become airborne again. Also, when water is applied to chrysotile it appears to prevent the fibres from becoming airborne, whereas crocidolite will dry out and become respirable again.[217]

Not only are crocidolite fibres more likely to become airborne and hence respirable in any given dust cloud, crocidolite is more likely to have a higher percentage of fibres of hazardous dimension than chrysotile or indeed, it may be added, than amosite.[218] To put this point in practical terms, it would appear that at any given fibre concentration as measured by the optical microscope, there are more likely to be a larger number of long, thin fibres in a crocidolite dust cloud than in either an amosite or chrysotile dust cloud. As between the latter two fibre types, the evidence suggests that amosite is more likely to generate a greater number of fibres of hazardous dimension than chrysotile.

Once inhaled, the needle-like shape of the amphiboles also likely allows for deeper penetration into the peripheral portions of the lung, pleural, and abdominal surfaces than chrysotile fibres which, because of their curliness, have a greater cross-sectional area, thereby increasing the opportunity for interception, decreasing the likelihood of penetration, and possibly decreasing movement after tissue penetration.[219]

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[216] RCA Exhibit II-57, Tab 8, in RCA Transcript, Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41: Ross Hunt, "The Asbestos Bundle Cleavage Difference Between Sheet Silicate (Chrysotile) and the Chain Silicate (Crocidolite)," Cleckheaton, Yorkshire, BBA Group PLC, Industrial Health Unit, 26 May 1982. (Mimeographed.) See also, W.H. Walton, "The Nature, Hazards and Assessment of Occupational Exposure to Airborne Asbestos Dusts: A Review," *Annals of Occupational Hygiene* 25 (1982): 117-247, as cited in Acheson and Gardner, *Asbestos: The Control Limit for Asbestos*, paragraph 37, p. 7; and Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, pp. 17-18.

[217] RCA Transcript, Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41, pp. 75-77, 84.

[218] RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, p. 96. Mr. Berry testified that the crocidolite fibres that tend to be distributed in the air are finer than the chrysotile fibres. Further, the long fibres of chrysotile are not completely straight, which means that aerodynamically they behave as if they are a lot thicker than the actual fibre diameter would suggest. See also, Walton, "The Nature, Hazards and Assessment of Occupational Exposure to Airborne Asbestos Dusts: A Review"; Chung-Yung Hwang and Graham W. Gibbs, "The Dimensions of Airborne Asbestos Fibres — I. Crocidolite from Kuruman Area, Cape Province, South Africa," *Annals of Occupational Hygiene* 24:1 (1981): 39; and Graham W. Gibbs and Chung-Yung Hwang, "Dimensions of Airborne Asbestos Fibres," in *Biological Effects of Mineral Fibres*, vol. 1, pp. 69-78.

These considerations provide a plausible explanation for the greater incidence of mesothelioma and for the fact that peritoneal mesothelioma occurs almost exclusively among amphibole-exposed populations; and also suggest that on balance the amphiboles are likely to be more hazardous than chrysotile. As between the amphiboles, in general crocidolite is likely to be somewhat more hazardous than amosite. Overall, there appears to be a correlation between fibre type and fibre dimension and thus at least to some degree a correlation between fibre type and the incidence of asbestos-related disease.

But precisely because the dimension of the fibre affects the incidence of disease, any asbestos fibres in the critical size range, no matter what the type, are likely to be hazardous. In other words, while the amphiboles may be more likely to generate airborne fibres of hazardous dimension, to the extent that particular industrial processes can generate finer or thinner chrysotile fibres, we would expect the hazardous nature of chrysotile exposure to increase. And that appears to have taken place. For when account is taken of the dimension of the fibre used in industry, the differences in the incidence of disease between mining and manufacturing, and, within manufacturing, as between textiles and friction products, become more understandable. For example, it is now widely thought that long, thin fibres are far more prevalent in textile operations than in other kinds of industrial or mining operations. The vigorous activity involved in certain of the textile operations, especially spinning and carding, tends to cause the fibres to split longitudinally, thus increasing the absolute number of fibres and making each thinner in diameter.[220] Chrysotile, which has a greater tendency to separate into fibrils than amphiboles, is likely to be particularly affected in this manner by textile operations.[221] In the manufacture of friction products nothing of this kind appears to take place. Rather, the fibres are ground together, causing them to break horizontally, thereby making them shorter in length but not changing their diameter.[222]

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[219] Peto, Seidman, and Selikoff, "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment," p. 134; RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 45; Paul Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," in *Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods*, p. 136; R.L. Zielhuis, rapporteur, *Public Health Risks of Exposure to Asbestos*, Report of a Working Group of Experts, prepared for the Commission of the European Communities (Oxford: Pergamon Press, 1977), p. 10.

[220] RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, pp. 46, 112; RCA Transcript, Evidence of Dr. John M. Dement, 23 October 1981, Volume no. 32, p. 37; RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 41–42; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 58.

[221] RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 41–42. Dr. A.D. McDonald testified that the carding and weaving of chrysotile textiles fragment chrysotile as much as that fibre can be fragmented.

In fact, Dr. Dement and his colleagues investigated the characteristics of the airborne fibres at Charleston, using transmission electron microscopy, and found that 10 to 27% of all airborne fibres were longer than 5 microns in length. Moreover, compared with friction product and cement product operations using chrysotile, textiles were found to have a substantially higher fraction of the long, thin fibres that Stanton and others have implicated as being most carcinogenic.[223] A similar finding was made by Dr. Graham W. Gibbs who testified before us that the proportion of long fibres in certain areas of textile manufacturing, like spinning, appears to be substantially more than in mining. He noted that the mining industry produces a whole variety of different grades of fibres. However, in the secondary industries such as textiles, it is only longer fibres that appear to be utilized.[224] In a subsequent study of the dimension of different fibres in a variety of industrial processes, Gibbs and Hwang have shown that in terms of diameter alone, chrysotile fibres used in textile carding operations are potentially more respirable than chrysotile or other fibres at different stages of processing.[225]

These findings assist in explaining the otherwise startling difference in disease incidence between the textile factory at Charleston, on the one hand, and, on the other, the friction plants at Derbyshire (the Ferodo plant) and Connecticut and the asbestos-cement factory at Cardiff. These findings, however, also clearly demonstrate that, subjected to certain processing, chrysotile is quite capable of producing fibres of very hazardous dimension.

A consideration of the physical parameters of asbestos fibres also appears to reconcile the observed incidence of mesothelioma in and around the Cape crocidolite mines in South Africa and the apparent absence of this tumour among the crocidolite and amosite miners of the Transvaal. Timbrell, Griffiths, and Pooley, in comparing the fibre dimensions from these two regions, ascertained that: (i) crocidolite fibres from the Cape are finer by a factor of 3; (ii) due to their larger diameter, the free-falling speed of Transvaal fibres is 9 times that of Cape fibres, meaning that Transvaal fibres are less likely to become airborne and will settle more rapidly; and (iii) fibres from the Transvaal have a greater aerodynamic size and thus a greater likelihood of interception in the larger airways of the tracheal-bronchial tree and less likelihood of penetrating to the periphery of the lungs.[226]

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[222] Zielhuis, *Public Health Risks of Exposure to Asbestos*, p. 22. Data compiled for this report indicated that the average fibre length in textile operations is between 8 and 24 mm; in asbestos-cement operations, between 2 and 13 mm; and in friction materials operations, between 2 and 4 mm. See also, RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, p. 112; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 58.

[223] Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," p. 883. (Discussion.) Dr. Dement indicated that on the basis of a deposition model used in their investigation, it was estimated that 13 to 15% of the airborne particles longer than 5 microns in length would be deposited in deep, non-ciliated portions of the lung.

[224] RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 125.

[225] Graham W. Gibbs and Chung-Yung Hwang, "Physical Parameters of Airborne Asbestos Fibres in Various Work Environments — Preliminary Findings," *American Industrial Hygiene Association Journal* 36:6 (June 1975): 464.

Further confirmation that the different health risks in mining are a function of fibre dimension comes from the work of Gibbs and Hwang who have compared the dimensions of fibres collected in the chrysotile mines in Quebec, the amosite mines of the Transvaal, and the crocidolite mines of the Cape.[227] Crocidolite fibres, because of their very thin diameter and relative length, tended to have a higher aspect ratio (length to diameter ratio) than either amosite or chrysotile. Amosite fibres, although thicker in diameter than the other two fibre types, were also longer and thus tended to occupy a middle position in terms of aspect ratio. Chrysotile fibres had the lowest aspect ratio of all. If it is assumed that fibres observed by optical microscopy are greater than 5 microns in length and greater than 0.3 microns in diameter, the percentage of total airborne fibres observed by Gibbs and Hwang during the bagging of crocidolite was 0.6%; of amosite, 16.9%; and of chrysotile, 1.6%. Conversely, if it is assumed that fibres greater than 5 microns in length and less than 0.3 microns in diameter are most hazardous, the number of fibres in that size range for concentrations of 2 f/cc, as measured by the optical microscope, would be 23 f/cc for crocidolite, compared to 0.9 f/cc for amosite, and 3 f/cc for chrysotile. Clearly, the results of this study in terms of fibre dimension are quite consistent with the observed incidence of mesothelioma among the three mining regions.[228]

The studies by Gibbs and Hwang also offer an explanation for the high incidence of disease among North American insulation workers exposed to amosite. While the percentage of long, thin fibres in dust clouds at the Transvaal mines appeared to be quite low and was thus consistent with the apparent absence of asbestos-related disease from these mines, the diameter of these fibres tended to decrease considerably from the raw amosite dumping stage to the stage when it was to be applied to building surfaces for insulation.[229] Indeed, the authors' results suggested that at this stage amosite dust concentrations had a slightly higher percentage of fibres greater than 5 microns in length and less than 0.5 microns in diameter than raw crocidolite and a much higher percentage of fibres of these dimensions than chrysotile in textile carding operations.[230]

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[226] V. Timbrell, D.M. Griffiths, and F.D. Pooley, "Possible Biological Importance of Fibre Diameters of South African Amphiboles," *Nature* 232 (2 July 1971): 55–56. The mean fibre diameters for the samples used by the authors were as follows: North Western Cape crocidolite, 0.073 microns; Transvaal crocidolite, 0.212 microns; and Transvaal amosite, 0.243 microns. The free-falling speed of fibres (a measure of particle aerodynamic size) is nearly proportional to the diameter squared and increases only slowly with an increase in length.

[227] Gibbs and Hwang, "Dimensions of Airborne Asbestos Fibres."

[228] Ibid., p. 76.

What these observations all tend to indicate is that there is indeed an association between type of industrial process and fibre dimension, and because of this, there is accordingly an association between industrial process and disease. A consideration of fibre dimension thus serves to assist in explaining the differing disease incidence in various industrial cohorts and has served to assist in explaining the differing incidence at least in mesothelioma among asbestos workers exposed to different fibre types. In our judgement, a consideration of fibre dimension also serves to reconcile the otherwise conflicting animal and epidemiological evidence on fibre type.

The most successful experiments in producing malignant tumours have been injection experiments. But these experiments do not take account of the importance of fibre dimension in terms of the respirability of the fibres and, once inhaled, in terms of their capacity to reach the site of tumour production. Moreover, most animal experiments have tended to use fibres of dimensions not found in industry. The UICC Reference Samples, including those for chrysotile, are all much finer in diameter than are those used industrially.[231] When the size distribution of the fibres used in animal experiments is taken into account, then the animal data and human epidemiological data become quite consistent. For example, in the large animal injection experiment reported on by Wagner, Berry, and Timbrell in 1973, UICC crocidolite with a higher proportion of long fibres produced more mesotheliomas than the UICC chrysotile.[232] Only the artificially separated superfine Canadian chrysotile appeared to be more hazardous. The recent inhalation study by Dr. Davis, in which chrysotile produced more fibrosis than the amphiboles and produced all of the malignant lung tumours, is readily explained as a matter of fibre dimension: 7% of all fibres in the chrysotile dust clouds were 20 or more microns in length, whereas in the amphibole dust clouds less than 0.5% of all fibres were greater than 20 microns in length.[233] *In vitro* tests also support the proposition that only longer fibres have a pathogenic effect on cell membranes, with fibres less than 5 microns in length being quickly and completely engulfed.[234]

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[229] Gibbs and Hwang, "Physical Parameters of Airborne Asbestos Fibres in Various Work Environments — Preliminary Findings," p. 464.

[230] Ibid., p. 465. The authors' study provided the following percentages of fibres with lengths greater than 5 microns and diameters less than 0.5 microns present in airborne dust: amosite application of insulation, 18.3%; crocidolite, 17.5%; amosite dumping, 17.0%; chrysotile dryer, 1.45%; chrysotile bagging, 1.0%; and chrysotile carding, 0.4%. The authors noted that if the combination of length greater than 5 microns and diameter less than 0.5 microns was the only factor responsible for the production of mesothelioma, their results would suggest that the hazard for the manufacturer or the insulation worker using amosite would be considerably greater than for chrysotile workers. Ibid.

[231] RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 29, 76-77. See also, Wagner et al., "The Effects of the Inhalation of Asbestos in Rats," p. 268; the authors observed, however, that there appears to be a trend for industry to use finer chrysotile, so that experimental results may be more relevant to current industry than to past.

Thus, we conclude from all of this evidence that the pathogenicity of asbestos is primarily a function of fibre dimension. We also conclude that the amphiboles, and especially crocidolite, are more likely to be of hazardous dimension than chrysotile. Moreover, the significance of fibre dimension in the production of disease has implications for fibre substitutes for asbestos. To the extent that man-made mineral fibres are long and thin in dimension, their use may also pose a health risk.[235] The health effects of substitutes for asbestos are considered in more detail in Chapter 6, Section E.

But while we believe that fibre dimension is the most biologically relevant characteristic of asbestos, we cannot totally discount the importance of other considerations. One such consideration is the chemistry and surface properties of asbestos fibres. Several studies published in the late 1960s and early 1970s have pointed to the possible importance of the fibre's surface chemistry in causing disease, although there is some disagreement as to which of the chemical characteristics of asbestos fibres are important.[236] There is, however, some evidence that the chemistry of chrysotile, and particularly its magnesium component, is such that it is more likely to dissolve in lung fluid over time. It is known that the amphiboles generally will not dissolve and thus may be likely to last longer in the lung than chrysotile.[237] And there is evidence to suggest that chrysotile fibres are cleared more readily from the lungs than are amphibole fibres.[238] If this were the case, it would provide an additional explanation for the apparently different results between the animal and human data insofar as chrysotile is concerned. It is possible that for all species, tumour induction requires a large fraction of a lifespan. It is possible that chrysotile may be able to survive in animals long enough to exert its full carcinogenic potential but may not always be able to do so in humans.[239]

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[232] J. Christopher Wagner, Geoffrey Berry, and Victor Timbrell, "Mesotheliomata in Rats after Inoculation with Asbestos and Other Materials," *British Journal of Cancer* 28:2 (August 1973): 173-185. See also, Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, p. 19.

[233] John M.G. Davis, "The Use of Animal Models for Studies on Asbestos Bioeffects," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 797.

[234] E.G. Beck, "Experimental Pathology — *In Vitro* Studies — Related to Asbestos and Other Mineral Fibres," in *Biological Effects of Mineral Fibres*, vol. 1, p. 389.

[235] For example, Stanton and Wrench found that glass fibres in the size range over 8 microns in length and less than 1.5 microns in diameter were almost as effective as asbestos in producing tumours after animal injection experiments. See Stanton and Wrench, "Mechanisms of Mesothelioma Induction with Asbestos and Fibrous Glass."

[236] Many of the studies suggesting the importance of the chemistry and surface properties of asbestos fibres are discussed in Jacques Dunnigan et al., "Cytotoxic and Haemolytic Effect of Native and Chemically Modified Chrysotile," *Fourth International Conference on Asbestos*, Turin, Italy: 26-30 May 1980, Preprints, vol. II (Turin, Italy: Istituto di Arte Mineraria del Politecnico & Associazione Mineraria Subalpina, 1980), pp. 747-772.

A group of epidemiologists from France who have been studying the health effects of asbestos place particular importance on the surface properties of asbestos as a determinant of disease. While they clearly admit the importance of fibre dimension in explaining how asbestos fibres reach the site of tumours, they also believe that once at the site, the interaction between the surface of the fibre and the surface of the cell has significance in disease production.[240]

Dr. Jacques Dunnigan of the Institut de recherche et de développement sur l'amiante at Sherbrooke, Quebec, testified before us that there is evidence that asbestos fibres become chemically altered or coated during processing and that these alterations may have the effect of making fibres less toxic.[241] Two examples he gave were the encapsulation of chrysotile fibres in resin in certain asbestos applications such as the manufacture of friction products and the calcium-coating that may chemically change chrysotile during asbestos-cement manufacturing.[242] In a similar vein, it has been suggested by some authorities that the milling process destroys the fibrous structure of chrysotile to a greater extent than the amphiboles.[243] Dr. Dunnigan and others are now engaged in research which seeks to alter the chemical characteristics of asbestos so as to render the fibres less toxic but at the same time maintain their useful properties.

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[237] RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, p. 67. Dr. Davis testified that it can be demonstrated that even in a weak acidic solution the magnesium ions can be leached out of the structure of a chrysotile fibre. One is still left with a fibril that looks much the same, but chemically it can be shown to have much less magnesium. Once the magnesium has left, the fibril is physically much weaker and more easily broken up. See RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 55-56.

[238] See Margaret R. Becklake, "Exposure to Asbestos and Human Disease," *The New England Journal of Medicine* 306:24 (17 June 1982): 1480-1482. The evidence that there is a preferential clearance of chrysotile as compared with amphibole fibres from the lung is largely based upon case-control lung tissue studies which indicate an excess of amphiboles in the lungs of the cases whereas the chrysotile content was similar in both cases and controls.

[239] Davis, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature,* p. 17.

[240] Personal communication between Professor Patrick Sebastien, Institute of Occupational Health and Safety, McGill University, Montreal, Quebec and Royal Commission on Asbestos Staff, 21 April 1983. See also, J. Bignon and M.C. Jaurand, "Biological *In Vitro* and *In Vivo* Responses of Chrysotile Versus Amphiboles," *Environmental Health Perspectives* 51 (September 1983): 73-80.

[241] See RCA Transcript, Evidence of Dr. Jacques Dunnigan, 20 August 1981, Volume no. 28, pp. 43-51, 83-88.

In our judgement, the chemistry and the surface properties of asbestos fibres have yet to be shown to be as important as fibre dimension in determining disease. However, in a subject that is bedevilled by uncertainty, we do not discount the fact that these characteristics of asbestos fibres may have a significant role to play. It may well be the case that future research will cast a clearer light on their importance. At this stage we draw sustenance from the fact that the evidence which does exist in the realm of chemistry and surface properties points to a similar conclusion as the evidence on fibre dimension: namely, that chrysotile tends to be less hazardous than the amphiboles.

There is one further and related consideration that also tends to suggest chrysotile may be less hazardous. This is its tendency to split apart continuously and ultimately to break up into tiny fibrils which might then be easily cleared from the lungs. Whether it is a matter of fibre chemistry or the physical tendency of chrysotile to split into tiny fibrils, there is considerable evidence that chrysotile fibres do not have the same long-term durability in the lungs as do the amphiboles.[244] This observation is consistent with the results of lung tissue studies which strongly suggest that there is a preferential clearance of chrysotile from the lungs as compared to the amphiboles.[245] To the extent that length of residence in the lung is important in disease production, this consideration too would implicate the amphiboles as more hazardous.

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[242] Ibid., pp. 43-48, 83-84. In his testimony, Dr. Dunnigan did not suggest that fibre dimension should be disregarded in assessing the toxicity of asbestos fibres, but rather that the chemical properties of fibres should be considered in addition to fibre dimension. RCA Transcript, Evidence of Dr. Jacques Dunnigan, 20 August 1981, Volume no. 28, p. 64. In his testimony, Dr. Weill noted that it had been suggested that asbestos in the asbestos-cement industry has a calcium-containing coating and that this coating may make it less pathogenic. See RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, p. 97.

[243] RCA Transcript, Evidence of Dr. Jacques Dunnigan, 20 August 1981, Volume no. 28, pp. 25-26. See also, K.R. Spurny, H. Opiela, and G. Weiss, "On the Milling and Ultrasonic Treatment of Fibres for Biological and Analytical Applications," in *Biological Effects of Mineral Fibres*, vol. 2, pp. 931-933.

These various considerations that we have discussed, particularly fibre dimension, appear to offer a reasonable explanation for the observed incidence of asbestos-related disease. But, in our judgement, a degree of uncertainty still remains. For example, we cannot totally dismiss the importance of short fibres in the production of disease. While experimental studies indicate that, once in place, longer fibres are more carcinogenic than short, it has been suggested that shorter fibres more easily migrate through the body and penetrate the tissue and thus more easily reach the sites of tumours.[246] It has also been suggested that due to the much larger number of short fibres, their overall effect may be greater than long fibres depending upon the size distribution of a given dust cloud, although this suggestion appears to be inconsistent with the engulfing activity of the macrophages.[247] A recent study by Professor Patrick Sebastien et al. analyzed the asbestos fibre content of tissue of the pleura from 29 cases exposed to mixed asbestos dusts and with a variety of tumours.[248] The surprising finding of this study was that virtually all of the fibres retained in the pleura were short chrysotile fibres, whereas the longer amphibole fibres were found in the lung parenchyma tissue. We cannot account for this finding if it suggests that short chrysotile fibres are largely responsible for mesothelioma production, since it is a finding which is contrary both to experimental work on fibre dimension and to the known incidence of mesothelioma in human populations.[249]

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[244] In his review article, *Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially Used Asbestos: A Review of the Literature*, at p. 7, Dr. Davis noted that the reason why most researchers have found relatively more amphibole fibres in human lung specimens, in spite of the fact that chrysotile makes up the bulk of industrial asbestos usage, was probably indicated by M.C. Jaurand et al. in 1976. In an article entitled "Solubilité du chrysotile *in vitro* et dans le poumon humain," *Revue française des Maladies Respiratoire* 4, Supp. 2 (1976): 111–120, the authors produced evidence of the chemical dissolution of chrysotile in the lung tissue. Dr. A.D. McDonald testified that amphibole fibres tend to remain as longer fibres once they get into body tissue, whereas chrysotile fibres tend to split up into much shorter pieces. See RCA Transcript, Evidence of Dr. Alison D. McDonald, 27 August 1981, Volume no. 31, pp. 47–48. See also, RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, pp. 131–132. In his testimony, Dr. Gibbs noted that amphibole fibres are very acid resistant and certainly not water soluble. Chrysotile, on the other hand, has been demonstrated to be not very acid resistant, and it has a magnesium hydroxide outer layer, so the possibility for leaching to occur does exist. But Dr. Gibbs also noted that the ability to break off a bundle is far greater for chrysotile than it is for amphibole fibres. This ability of chrysotile fibres to break apart might well improve their ability to be removed from the lung.

[245] See Becklake, "Exposure to Asbestos and Human Disease," and the lung tissue studies cited therein.

[246] See RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 47–48; RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 130; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, p. 71.

There is other uncertainty. For example, it is not entirely clear in our judgement why chrysotile is capable of producing an extraordinarily high incidence of lung cancer and yet only one mesothelioma has been found at Charleston. It may be that chrysotile fibres, even in the hazardous fibre dimensions that are likely found in textile operations, are still not able to migrate to the pleura or to the peritoneal cavities as efficiently as the amphiboles, but this is not known with certainty.

There are different cohorts of workers manufacturing the same products, apparently using the same processes and the same mix of fibre types, that still exhibit widely different health effects. For example, there is a substantial difference in the amount of asbestos-related disease between the Johns-Manville plant at Scarborough and the two asbestos-cement plants at New Orleans, a difference that persists even when account is taken of the selective exposure to crocidolite at the New Orleans factory. Neither Dr. Finkelstein nor Dr. Weill has been able to offer a plausible explanation for this difference.[250] It could be that the explanation might be found in climatic differences between Ontario and Louisiana and consequent differences in plant ventilation.

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[247] RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 47–48. Dr. Chatfield, in "Airborne Asbestos Fibres: A Summary of Some Measurement Problems," at p. 1, noted the work of Dr. Pott which indicated that although there was a declining carcinogenicity factor for a single fibre below 5 microns in length, it was still significant below this length. Dr. Chatfield went on to observe that since in airborne material there are many more short fibres than longer ones, the overall biological effect of the shorter fibres may actually be larger than that of the less numerous long fibres.

[248] Patrick Sebastien et al., "Asbestos Retention in Human Respiratory Tissues: Comparative Measurements in Lung Parenchyma and in Parietal Pleura," in *Biological Effects of Mineral Fibres*, vol. 1, pp. 237–246.

[249] This somewhat surprising finding was referred to by a number of witnesses who testified at our hearings. See RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 71–73; RCA Transcript, Evidence of Dr. Graham W. Gibbs, 21 August 1981, Volume no. 29, p. 130; and RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 126–127. In his testimony, Dr. Davis suggested the possibility that the cases studied by Sebastien et al. may have had long fibres of chrysotile in the pleura previously which had broken up over time. Another possibility is that the break-up could have taken place elsewhere within the body and that subsequently these now shorter fibres could have found their way to the pleura and remained there.

What this discussion amply illustrates is that the final chapter on the pathogenesis of asbestos-related disease is yet to be written. However, we believe that the conclusions that we have reached in this chapter fairly represent the current state of the evidence.

E. Dose-Response Relationships

E.1 The General Principle

The dose-response relationship is a general biological principle that refers to the correspondence between the extent of exposure (that is, the "dose") and the biological outcome of interest (the "response"). If one can demonstrate an increasing (or decreasing) risk of severity of response, for example, lung cancer, in association with an increased (or decreased) dose of exposure to the putative cause, for example, asbestos, then one can say there is a dose-response relationship.[251] The nature of that relationship will turn upon the shape and location of the line or curve when it is plotted on a graph. If, for example, an increase in the dose of asbestos leads to a proportional increase in the excess of lung cancer, then the dose-response relationship for lung cancer is said to be linear in nature and is described by a straight line. Shapes other than linear are clearly possible.

The significance of the concept of a dose-response relationship is at least twofold. First, it is a key element in establishing a causal relationship between the dose and the response as well as a tool for explaining variations in biological response. Second, in practical terms, it provides information upon which to establish regulatory standards. A linear dose-response relationship is particularly attractive for regulatory purposes because, once assumed, it permits by extrapolation a calculation of relative risk at exposure levels too low to be detected by normal epidemiological studies.[252]

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[250] Letter from Dr. Hans Weill, Science Policy Fellow, The Brookings Institution, Washington, D.C. to the Royal Commission on Asbestos, 13 July 1983.

[251] For a discussion of the dose-response relationship, see David L. Sackett, "A Review of Diagnostic Tests for Causation," in Ontario, Royal Commission on Asbestos, *Proceedings of The Royal Commission on Asbestos, Second Public Meeting, Friday, December 12, 1980*, reported by Lydia Dotto (Toronto: Royal Commission on Asbestos, 1981), pp. 13-14 and Appendix B, pp. 6-8.

[252] J.C. McDonald and Liddell, "Mortality in Canadian Miners and Millers Exposed to Chrysotile," p. 8.

E.2 A Threshold or "No-Effect" Level

Closely related to the concept of a dose-response relationship is the notion of a threshold: that is, a level of dose below which no effect or no risk of illness or death will occur. When one charts a dose-response relationship on a graph to ascertain whether or not a threshold exists, one should examine the location and contour of the dose-response relationship at the point of zero dose. If the response at the point of zero dose is zero (or positive), and the slope of the dose-response line or curve is positive (that is, a line going out from the origin), this suggests there is no dose at which some response will not occur. In other words, this suggests that no threshold exists. If, on the other hand, there is zero response at a dose greater than zero, this suggests that there are doses at which no change in response is expected; that is, a threshold or "no-effect" level exists.[253]

The existence or non-existence of a threshold for asbestos-related diseases has stirred considerable controversy among those who seek to legislate an acceptable level of exposure. If indeed a threshold level does exist, such a level poses no risk. If there is no threshold, then we must accept the proposition that whatever level of exposure is chosen as the regulatory control limit, it will always carry with it at least some risk of disease.

E.3 The Nature of the Dose-Response Relationships for Asbestos-Related Diseases

From the foregoing it is evident that the nature of the dose-response relationships for the asbestos-related diseases and the existence or non-existence of a threshold or no-effect level for these diseases are important from a regulatory standpoint. For ease of exposition, we discuss dose-response in relation to the three principal diseases separately.

(a) Asbestosis

The preponderance of data from the various working populations that have been studied — including the Quebec chrysotile miners, the Rochdale and Charleston textile workers, and the Scarborough asbestos-cement workers — clearly evidences a positive dose-response relationship for asbestosis.[254] And while we are told that biologically a linear dose-response relationship is less plausible for a chronic fibrotic process such as asbestosis than it is for cancer,[255] nevertheless the data are consistent with a linear relationship, and we are satisified that such a linear relationship appears to describe the results of the various studies satisfactorily.[256]

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[253] Sackett, "A Review of Diagnostic Tests for Causation," Appendix B, p. 8.

[254] See Section B of this chapter.

We are less satisfied that there is no threshold for the clinical manifestation of asbestosis. Indeed, there was a general consensus among the expert witnesses who appeared before us that mortality associated with asbestosis is related to high doses and that at the current much lower exposure levels found in industry even clinically disabling asbestosis is much less a matter of concern than it has been in the past.[257] Biologically this seems plausible. Unlike cancer, which may be initiated by a "trigger dose" of asbestos, it would seem that the clinical manifestation of asbestosis requires the inhalation of a considerable amount of asbestos over time. As Dr. J. Corbett McDonald testified, a person has ". . . to have fibrosis beyond a certain level before it could conceivably do anything to you, because the reserve power of the lung is fairly substantial. . . ."[258]

Viewed in this light, what is material about asbestosis involves not so much the existence or non-existence of a threshold for the initiation of fibrosis; what is more relevant is the relationship of asbestos exposure to the development of the fibrotic process. As we assess the epidemiological literature there is a growing body of data to suggest, if not confirm, that there is a low level of asbestos exposure below which the clinical manifestation of asbestosis will not occur, as distinct from the initiation of fibrosis which does not produce detectable changes in normal pulmonary function. One important indication of this fact is that the development of clinical asbestosis has been almost exclusively limited to occupationally exposed populations where exposures have historically been heavy.[259] There is no evidence that it has ever been caused by asbestos exposure outside the workplace. Even, for example, the extensive study of the household contacts of workers at the Paterson, New Jersey, amosite insulation factory conducted by Dr. Henry A. Anderson et al. revealed minimal parenchymal abnormalities.[260] A total of 679 family members who had lived with workers at the plant during their employment (1941–1954) were all examined 20 or more years after the time of their first exposure.[261] They were essentially healthy when examined; pleural changes predominated and very few had even advanced irregular opacities.[262]

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[255] See J.C. McDonald and Liddell, "Mortality in Canadian Miners and Millers Exposed to Chrysotile," p. 8. See also, RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 33-34; and RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, p. 29. See also, Peto, "The Hygiene Standard for Chrysotile Asbestos," at p. 488, where the author has stated that "There are no grounds for assuming linear dose-response for such a generalised progressive disease . . . ."

[256] For example, in his own studies, Dr. J.C. McDonald testified that he finds a linear relationship for both asbestosis and lung cancer and also, although not as good, for gastrointestinal cancer. See RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 33-34. Dr. Acheson in his testimony noted that a linear hypothesis fits the data from Rochdale and from New Orleans. See RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, pp. 14-15.

[257] Dr. Finkelstein testified that ". . . for all practical purposes there is probably an effective threshold below which clinical asbestosis will be unlikely to occur, although there might be the occasional very susceptible individual who could develop problems." See RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 70. Dr. Nicholson testified that "There really is like a threshold for mortality from asbestosis." See RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 66. See also, Peto, "The Hygiene Standard for Chrysotile Asbestos," p. 488; and RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, p. 96.

[258] RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 34.

The fact that there have been no cases of asbestosis among those not occupationally exposed to asbestos indicates that the fibrotic process does not progress to the point of clinical manifestation among those with low exposure. We note that the absence of clinical asbestosis among the general public led the U.K. Advisory Committee to suggest ". . . that there may be a threshold level here below which asbestosis is not detectable."[263]

If those not occupationally exposed to asbestos have no risk of developing clinical asbestosis, what is the risk for those occupationally exposed? Dr. E. Donald Acheson and Dr. Martin J. Gardner, in their report to the U.K. Advisory Committee, "The Ill Effects of Asbestos on Health," came down in favour of a dose-response relationship without a threshold for chrysotile asbestos within the range of exposures experienced in industry.[264]

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[259] Dr. Finkelstein testified: "I think that asbestosis is not a public health problem . . . . I believe that asbestosis is almost exclusively limited to occupationally exposed populations. That's clinical asbestosis." See RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 71.

[260] Henry A. Anderson et al., "Asbestosis Among Household Contacts of Asbestos Factory Workers," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 387–399.

[261] Ibid. The study was limited to the 679 household contacts who had lived in a household of an actively employed amosite asbestos factory worker and who themselves had not had an occupational exposure to asbestos or other fibrogenic dust.

[262] See RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, pp. 39–40. Dr. Anderson admitted that while some of these family members are at increased risk, still the likelihood for any one of them to reach the point of disability from asbestos exposure is low.

[263] U.K., Advisory Committee on Asbestos, *Asbestos — Volume I: Final Report of the Advisory Committee*, paragraph 103, p. 58.

[264] Acheson and Gardner, "The Ill Effects of Asbestos on Health," paragraph 191, p. 38. In his evidence before this Commission, Dr. Acheson testified that "One may be less certain that there is no threshold in relation to asbestosis, in view of the fact that although it is known that a very large proportion of the urban population of industrialized Europe and North America have substantial numbers of asbestos fibres in their lungs, they do not at autopsy have evidence of asbestosis. So it may very well be that there is a threshold in respect of asbestosis." RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 15.

However, at the time their report was written, the control limit for chrysotile asbestos in the United Kingdom was 2 f/cc and the calculation of health risks was predicated on a working life of 50 years, yielding a cumulative dose of 100 f/cc-yrs.[265] What is the likelihood of clinical asbestosis occurring when the cumulative exposure is one-half, one-quarter, or one-fifth of this level?

Dr. Finkelstein has recently examined the cumulative probability of having developed certified asbestosis (that is, being adjudged by the Ontario Workers' Compensation Board to have asbestosis) by the end of the 32nd year from first exposure among 157 long-term employees (15 or more years' employment) of the Scarborough Johns-Manville plant.[266] He found the probability to be 90% among the very heavily exposed, that is, with a cumulative exposure in the range of 200 to 249 f/cc-yrs; falling to 10% among men with a cumulative exposure of less than 50 f/cc-yrs; and to 1% or less around 10 f/cc-yrs. (See Figure 5.1.) These results led Dr. Finkelstein to conclude that ". . . the major risk at lower exposures will be due to cancer rather than to asbestosis."[267]

Similar findings to those of Dr. Finkelstein were made by Mr. Berry in his examination of 197 workers employed after 1950 and for 10 or more years at Rochdale.[268] Here the average follow-up was shorter, averaging 16 years, but Mr. Berry was looking only at the prevalence of "possible asbestosis," essentially a combined judgement of two physicians as to whether a worker had developed signs attributable to early asbestosis. Average annual exposure for the 197 workers was 5 f/cc and the prevalence of possible asbestosis, 6.6%. This prevalence rate fell to zero or near zero at cumulative exposures of 10 f/cc-yrs.[269]

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[265] British Occupational Hygiene Society, Committee on Hygiene Standards, "Hygiene Standards for Chrysotile Asbestos Dust," pp. 51–52. Using basal rales as the key symptom, the authors of the report concluded that the concentration giving a risk of 1% for an individual working for 50 years in the asbestos industry was estimated to be 2.2 f/cc as measured with the standard membrane filter method.

[266] Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," pp. 496–501.

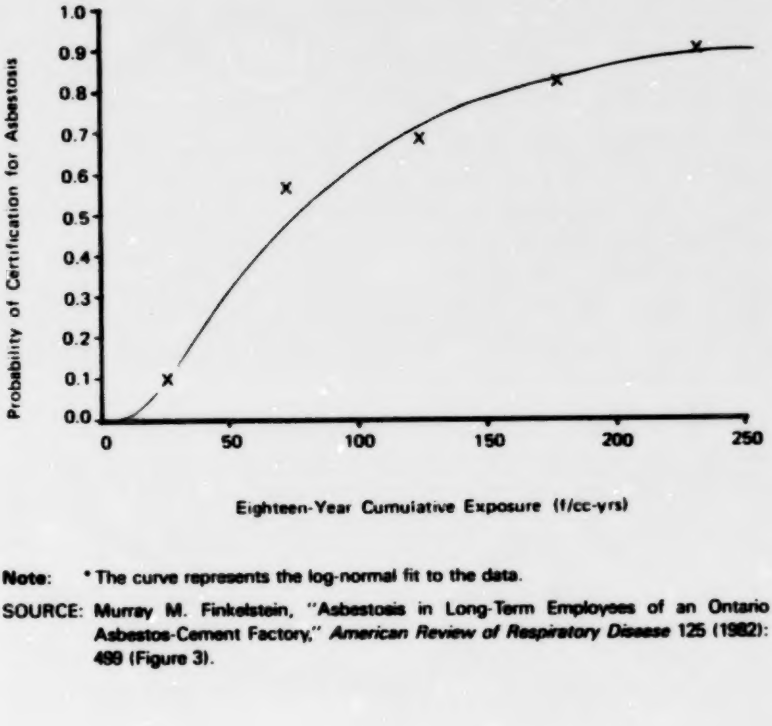
[267] Ibid., p. 501.

[268] Berry et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory."

[269] Ibid., p. 109. The authors noted that the average cumulative exposure of the group first employed after 1950 at Rochdale was 84 f/cc-yrs. In view of their findings, the authors concluded that ". . . there is no room for complacency about the 2 f/cm^3 standard . . . ." The authors also presented data as to the prevalence of certified asbestosis in relation to cumulative exposure. Once again, the average interval from first exposure was 16 years and the maximum was 23 years. For workers in the 50 to 99 f/cc-yrs category, they observed a prevalence rate for certified asbestosis of about 2.5% and for workers in the 100 to 149 f/cc-yrs category, the prevalence rate was 8.5%. By comparison, Dr. Finkelstein's probabilities, evaluated at the end of the 22nd year from first exposure, were 4% and 6% respectively. See Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 501.

Figure 5.1

The Cumulative Probability of Certification for Asbestosis (as evaluated at the end of 32 years from first exposure) Versus the 18-Year Cumulative Exposure*



Note: * The curve represents the log-normal fit to the data.

SOURCE: Murray M. Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," *American Review of Respiratory Disease* 125 (1982): 499 (Figure 3).

An even more recent study of Rochdale has been conducted by the British Occupational Hygiene Society (BOHS). In updating Mr. Berry's work, the BOHS Committee on Asbestos examined 295 men who had commenced employment after 1950 and who had a minimum of 10 years' exposure to asbestos by December 1976.[270] However, the Committee's end point was not "certified asbestosis" or even "possible asbestosis." It was instead whether one of seven defined radiological or clinical adverse effects was present.[271] The Committee found that the probability of any one of these effects occurring at exposures less than 50 f/cc-yrs was 7%, falling to under 2% for cumulative exposures less than 25 f/cc-yrs.[272]

Dr. Weill's earlier findings from his examination of 859 workers employed in two asbestos-cement manufacturing plants in the New Orleans area are also indicative of a cumulative exposure level below which clinical asbestosis would be unlikely to occur, at least in that cohort. Using a conversion ratio of 2 f/cc for 1 mppcf, Dr. Weill suggested that below 100 mppcf-yrs, or 200 f/cc-yrs, diffuse pulmonary fibrosis would not likely be found. Such a level would be equivalent to an exposure of 5 f/cc per year for a working lifetime of 40 years; or an exposure of 8 f/cc per year for a working lifetime of 25 years.[273]

We note also Dr. Enterline's observation that despite the many workers exposed to asbestos in the United States, there have been very few reported deaths from asbestosis since 1950; in fact, between 1950 and 1977, there were 747 reported deaths from asbestosis, with the trend rising until 1972 and then levelling off at an average of approximately 50 per year.[274]

We acknowledge that these data do not establish beyond any doubt the existence of a cumulative exposure level below which clinical asbestosis will not occur. We recognize that among some cohorts studied, even workers in the lower cumulative exposure categories (which correspond more closely to modern conditions) have died as a result of asbestosis. We recognize too that variations in susceptibility among individuals make it difficult to have any confidence in a no-effect or threshold level. What is a threshold for one person may well not be a threshold for another. Nevertheless, our assessment of the evidence finds us in agreement with Dr. Finkelstein's comment that there is probably an effective threshold below which clinical asbestosis may be unlikely to occur, although there may be occasional cases developing in very susceptible individuals.[275] At low levels of occupational exposure to asbestos the fibrotic process in the lungs, if indeed it can be initiated, will not likely progress to the point of clinical manifestation or even the mildest discomfort. On the basis of the available data, our best judgement as to the lifetime occcupational exposure to asbestos at which the fibrotic process cannot advance to the point of clinical manifestation of asbestosis is in the range of 25 f/cc-yrs and below. We are sustained in this view by Dr. Weill's evidence to the effect that as a practising chest physician he now rarely sees asbestosis to a degree which is so functionally severe that it has an important impact on the cardio-respiratory system.[276] In our judgement, asbestosis can be deemed a disease of past high exposure levels and will not occur in workers exposed to the regulated levels of occupational exposure now in force in Ontario.

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[270] British Occupational Hygiene Society, "Report from the Committee on Asbestos: A Study of the Health Experience in Two U.K. Asbestos Factories," S.A. Roach, Chairman, *Annals of Occupational Hygiene* 27:1 (1983): 1–55.

[271] Ibid., p. 10, where the seven criteria are set out.

[272] Ibid., p. 1.

[273] See Weill et al., "Lung Function Consequences of Dust Exposure in Asbestos Cement Manufacturing Plants." See also, Hans Weill, "Radiographic and Physiologic Patterns Among Workers Engaged in Manufacture of Asbestos Cement Products: A Preliminary Report," *Journal of Occupational Medicine* 15 (1973): 248–252.

[274] Philip E. Enterline, "Proportion of Cancer Due to Exposure to Asbestos," in *Banbury Report 9: Quantification of Occupational Cancer*, Table 1, p. 21. In his testimony before the Commission, Dr. Enterline observed that at today's much lower exposure levels, clinically disabling asbestosis is much less a matter of concern than it has been historically. See RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, p. 96. Dr. Enterline also observed that in North America there is a long history of reporting of asbestosis cases, so it is unlikely that very many cases have gone unreported. RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, p. 9.

(b) Lung Cancer

Most epidemiological studies of asbestos workers that have demonstrated an excess lung cancer risk associated with the inhalation of asbestos have produced results consistent not only with a linear relationship between cumulative dose and mortality, but also consistent with the absence of a threshold. We include among these studies that of the Quebec chrysotile miners by Dr. J.C. McDonald et al.; the Charleston, South Carolina, textile workers studied separately by Dr. Dement et al. and Dr. A.D. McDonald et al.; and the Manville factory workers studied by Dr. Enterline.[277] In all of these studies there appears to be a progressive and proportional increase in the SMR for lung cancer with increasing dose and no evidence of a threshold level.[278]

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[275] RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, pp. 70–71.

[276] RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, p. 43.

[277] See the review of the various studies of asbestos workers in Section B of this chapter. Dr. Acheson testified that from his review of the evidence on lung cancer mortality, the data were consistent with a linear hypothesis and no threshold. See RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, pp. 14–15. Dr. Nicholson testified that none of the studies gives any evidence for a threshold effect of asbestos exposure and all of the studies are best described by the use of a linear dose-response relationship, although in some cases an even more severe relationship, that is, one that would have increased effect at lower exposure, may obtain. See RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 61.

This evidence cannot be accepted without some qualification, however, at least in part because of the limitations of the available data base. All of the studies have the intractable difficulty of separating out the effects of cumulative dose from duration of exposure.[279] Mr. Peto has even suggested that the apparently very good correlation between relative risk and cumulative dose found in many published studies actually reflects the correlation between duration of exposure and relative risk, and he points to his own recent study of the Rochdale textile workers as supportive of that proposition.[280] Moreover, there are some studies — for example, the Connecticut friction materials plant only just reported on by Dr. A.D. McDonald and the Scarborough cement workers reported on by Dr. Finkelstein — in which the higher lung cancer mortality rates are found not in the higher but in the lower cumulative exposure categories.[281] Such results are difficult to interpret but may reflect the uncertainty of the available data, including exposure estimates, the possible confounding effects of smoking, and selectivity in employment; or they may simply be attributable to chance.

Another study — Dr. Weill's New Orleans asbestos-cement workers — has produced results that are consistent both with and without the postulation of a threshold.[282] Indeed, in his evidence to this Commission, Dr. Weill suggested that an exposure level that was low enough to prevent asbestosis would likely be sufficiently low to preclude detectable excess risk for lung cancer.[283] By implication a threshold level for asbestosis might also be a threshold level for lung cancer. Both Mr. Berry and Dr. J.C. McDonald lent support to Dr. Weill's proposition, agreeing that if dust levels were reduced so that asbestosis did not occur then lung cancer would be mainly controlled, although both injected the caveat that it would not be completely controlled and some lung cancer cases would occur.[284]

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[278] In "The Hygiene Standard for Chrysotile Asbestos," Mr. Julian Peto observed that a linear relationship for lung cancer is supported by published work, and he referred both to Dr. Enterline's and Dr. J.C. McDonald's data. He noted that, by contrast, "If the duration of brief exposure can be taken as a measure of total dose the results of [the amosite study by Seidman, Lilis, and Selikoff] appear to suggest that for respiratory cancer lower doses may be proportionately more dangerous . . . ." (p. 486.) Such apparent non-linearity could be due to selective removal of workers who develop respiratory symptoms, saturation at very high exposures, underestimation of expected numbers, or random error in estimates of dose or duration of exposure. In summing up the various papers on asbestos delivered at the IARC conference in Lyon in 1979, Dr. Weill stated: "In regard to carcinogenic effects, there appeared to be a consensus that the shape of the curve is linear and that no threshold exists." See Hans Weill, "Asbestos — A Summing-Up," in *Biological Effects of Mineral Fibres*, vol. 2, p. 872.

[279] See RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 38–40; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 27.

[280] RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 137. See also, Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," p. 833.

[281] See Sections B.2 and B.4 of this chapter.

[282] Weill, Hughes, and Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing." See also, RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 75–77.

We do not find it surprising that there is debate about the carcinogenic risk at low exposure levels because lung cancer risks at low doses over a working lifetime have not to date been estimated by observation but rather by extrapolation from observed risks at higher exposure levels.[285] Accordingly, there is no direct evidence of the existence or absence of a threshold for lung cancer. It may arguably be the case that with further inquiry and better information the scientific community will be able to demonstrate that there is a dose level for asbestos for which the body's defence mechanisms are effective or that asbestos acts differently at lower rather than higher doses, thus demonstrating a threshold level for the induction of cancer. At the present time that information does not appear to us to exist. Since a threshold dose level for asbestos-related lung cancer has not been established, we believe it prudent to assume for regulatory purposes that there is none and that any dose may induce lung cancer.[286] This is certainly consistent with the weight of the epidemiological evidence. Several studies, for example, those by Dr. Dement and by Dr. Seidman, clearly show an excess risk of lung cancer mortality at concentrations where asbestosis is not an important cause of mortality.[287] Moreover, our assessment of the epidemiological literature is fortified by two further observations. First, from our understanding of the medical literature, a linear dose-response model without a threshold is biologically plausible and is consistent with current models of carcinogenesis.[288] In this vein, we note Dr. J.C. McDonald's testimony that the dose-response line for lung cancer is one of the most precise estimates of exposure-response in cancer published in any field today.[289] Second, a linear non-threshold model is less likely to underestimate the risks at low doses than any other plausible model.[290] In light of these observations and the strength of the epidemiological evidence, we have no hesitation in proceeding on the basis that there is a dose-response relationship for lung cancer and that it is best described by a linear non-threshold model or, on a graph, by a straight line through the origin. Of course, the slope of that line, or the degree of increase in relative risk for corresponding increases in dose, has varied widely among the different occupational cohorts that have been studied.

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[283] RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 34–35, 132.

[284] RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 41; and RCA Transcript, Evidence of Mr. Geoffrey Berry, 19 June 1981, Volume no. 11, pp. 83–84.

[285] Weill, "Asbestos — A Summing-Up," p. 872.

[286] In his article, "Dose-Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards, Part II. Mortality," at pp. 196–197, Mr. Peto suggested that there are three qualitative alternatives: (i) assume that because no excess risk has been demonstrated at levels below, for example, 2 f/cc, one should assume that such levels are virtually safe; (ii) assume that low levels are almost as dangerous as moderate levels until they are proved not to be; or (iii) assume a linear dose-response relationship and legislate accordingly. Mr. Peto rejected the first alternative as too optimistic and the second as too pessimistic and neither supported by biologic knowledge nor epidemiological data. He argued for the third alternative as being the most reasonable and as being a pragmatic proposal, rather than a scientific statement. He suggested that a linear dose-response relationship should be assumed until it is disproved for the purpose of setting a hygiene standard for asbestos rather than to argue that the theories of carcinogenesis that predict non-linearity are necessarily wrong.

[287] See Sections B.3 and B.5 of this chapter. See also, Seidman, Selikoff, and Hammond, "Short-Term Asbestos Work Exposure and Long-Term Observation," Table 3B, p. 65.

(c) Mesothelioma

The nature of the dose-response relationship for mesothelioma has been less firmly established than that for either asbestosis or lung cancer.

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[288] See Kenny S. Crump et al., "Fundamental Carcinogenic Processes and Their Implications for Low Dose Risk Assessment," *Cancer Research* 36 (September 1976): 2973-2979. In the article, the authors demonstrated that ". . . under some reasonable assumptions about carcinogenic mechanisms and processes that dose responses will be approximately linear at low doses." (p. 2977.) The authors went on to state that "Virtually all models of carcinogenesis that depict the exposure as affecting an already ongoing process will lead to linearity at low dose . . . . This result then implies that no matter what the biological mechanism we might imagine, if the carcinogen increases some part of the already ongoing process, then we should expect a response to be approximately linear at low dose." (p. 2978.)

[289] RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 14. Dr. J.C. McDonald also testified:

> . . . if we imagine that one component of the onset of malignant disease is damage to the nucleus to produce a mutation, each insult to the nucleus of cells, in this case lung cells, will carry, if you like, a finite risk that something will go wrong. So it is reasonable that the dose should be directly related to the probability of this mutation occurring. This will hold up even if there is, if you like, a repair mechanism, because the repair mechanism will still, if you like, have to be dealing with a hazard which is dose-related. (See RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 33.)

[290] RCA Transcript, Evidence of Dr. Kenny S. Crump, 13 August 1981, Volume no. 26, p. 33; and RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 13. Mr. Berry has noted that there are in addition two main practical objections to basing a hygiene standard on the assumed existence of a threshold: first, the value of this threshold could never be determined from the epidemiological data (Mr. Berry cited by way of example, if at a certain dose there were no cases of disease out of 1,000 individuals at risk, then it could only be stated with reasonable certainty that the risk was less than 3 in 1,000); and second, even if the threshold value were known, it would not be possible to conclude from a limited number of dust measurements that a worker would never be exposed to higher concentrations. See Geoffrey Berry, "Hygiene Standards — Theory and Application," in *Biological Effects of Asbestos*, pp. 145-149. See also, RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, p. 95.

Indeed, it has been suggested that very trivial doses of asbestos (even, some have said, no more than one day's exposure) are capable of inducing the disease and that as a result there is no dose-response relationship for mesothelioma at all.[291] That mesothelioma is associated with low levels of exposure for brief periods of time appears to be based upon isolated anecdotal case reports and upon more systematic case-series reports of mesothelioma arising from non-occupational household or neighbourhood exposures.[292] For example, Dr. Anderson, in his continuing investigation of disease among the household contacts of the amosite asbestos workers at Paterson, New Jersey, has already found 5 cases of pleural mesothelioma, and tracing of these contacts is not yet complete.[293] Dr. Anderson has also indicated that by 1979 there were published reports of over 50 mesothelioma deaths arising from domestic exposure.[294] He has further noted that in those cases arising in non-occupationally exposed individuals, pleural mesotheliomas predominated over peritoneal mesotheliomas.[295]

In the mesothelioma survey conducted by Dr. J.C. McDonald and Dr. A.D. McDonald, embracing 22 countries in the years 1959–1976, the authors identified 230 cases attributable to home or neighbourhood exposure.[296] We are of the opinion that this evidence is not inconsistent with the existence of a dose-response relationship for mesothelioma.

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[291] See RCA Transcript, Submission by Mr. Roy Steinfurth on behalf of the International Association of Heat and Frost Insulators and Asbestos Workers, 8 June 1981, Volume no. 7, p. 44; and Canadian Environmental Law Association, Written submission to the Royal Commission on Asbestos, #45, 17 February 1981, p. 9. In his testimony, Dr. Finkelstein referred to the fact that "One often hears quoted or sees in print the statement that because, you know, someone with one day's exposure has developed mesothelioma, there is no dose-response and this is hazardous at all levels." See RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 91. The witness rejected that view in his testimony. See note 308, *infra*. Mr. Berry testified that "Mesotheliomas are known to occur after incidental exposure, and this has led to several people putting forward the opinion that there was no dose-response relationship." See RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, p. 17. In fact, as Mr. Berry subsequently explained in his testimony, the first data establishing a dose-response relationship for mesothelioma were taken from a report by Newhouse and Berry on the Cape Asbestos East London factory. See Newhouse and Berry, "Patterns of Mortality in Asbestos Factory Workers in London."

[292] See RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 92–94. See also, Margaret R. Becklake, "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," *American Review of Respiratory Disease* 114:1 (July 1976): 211.

[293] Anderson et al., "Asbestosis Among Household Contacts of Asbestos Factory Workers," p. 387.

[294] Henry A. Anderson et al., "Household Exposure to Asbestos and Risk of Subsequent Disease," in *Dusts and Disease*, p. 155.

[295] RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, p. 20.

[296] J. Corbett McDonald and Alison D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," *Preventive Medicine* 6:3 (September 1977): 428–429.

The number of cases occurring among those either occupationally or non-occupationally exposed ought not to be considered without also having regard to the exposure levels involved and the number of persons at risk. For example, the 230 cases identified by the McDonald and McDonald survey occurred among a huge population at risk.[297]

More recent data which have considered these factors strongly support the existence of a dose-response relationship for mesothelioma, a relationship which, like that for lung cancer, is best described by a linear non-threshold model. The first such data came from the Newhouse and Berry study of the Cape Asbestos East London factory where approximately 7% of worker deaths were attributable to mesothelioma.[298] The authors, by dividing their cohort into four rough exposure categories of "severe" or "low to moderate" with more or less than 2 years' exposure, were able to demonstrate a dose-response relationship. (See Table 5.31.) Similarly, the study by Jones et al. of the female gas mask assemblers at Nottingham, England, in World War II (using crocidolite) showed that 2.8% of those workers with 10 to 20 months' exposure contracted mesothelioma, rising to 4.6% for those with 20 to 30 months' exposure, and to 9.8% for those with over 30 months' exposure.[299] In Dr. Finkelstein's mortality study of the Johns-Manville plant, while the numbers are admittedly small, there is a clear trend to an increasing risk of mesothelioma with increasing exposure and the results are compatible with a linear dose-response relationship.[300] We observe also that both animal studies and lung tissue studies evidence that mesothelioma operates in accordance with recognized principles of dose-response.[301] And as both Dr. Paul Kotin and Dr. William J. Nicholson

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[297] Ibid. The following countries were included in the study: Australia, Belgium, Canada, Cyprus, Czechoslovakia, Eire, Finland, France, Federal Republic of Germany, German Democratic Republic, Greece, Israel, Italy, Netherlands, South Africa, Spain, Sweden, Switzerland, U.K., U.S.A., U.S.S.R., and West Indies.

[298] Newhouse and Berry, "Patterns of Mortality in Asbestos Factory Workers in London."

[299] See Jones et al., "The Consequences of Exposure to Asbestos Dust in a Wartime Gas-Mask Factory," Table 5, p. 646.

[300] See Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," p. 18. See also, Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," p. 142.

[301] Dr. Davis testified that in his animal injection studies he gets very good dose-response relationships for mesothelioma. See RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, p. 95. See also, RCA Transcript, Evidence of Mr. Geoffrey Berry, 19 June 1981, Volume no. 11, pp. 28–29. See also, Wagner, Berry, and Timbrell, "Mesotheliomata in Rats After Inoculation with Asbestos and Other Materials." The authors, at p. 178, indicated that a varying dose experiment gave results which indicated that the risk of developing mesothelioma at a given time after injection was proportional to the dose. See also, F. Whitwell, J. Scott, and M. Grimshaw, "Relationship Between Occupations and Asbestos-Fibre Content of the Lungs in Patients with Pleural Mesothelioma, Lung Cancer, and Other Diseases," *Thorax* 32 (1977): 383.

Table 5.31

Mesothelioma Death Rates

Exposure Category and Duration (Years)	Pleura	Peritoneum	Subject-Years	Rate per 100,000 Subject-Years
Males				
Low to moderate				
<2	3	1	12,031	33
>2	3	4	7,500	93
Severe				
<2	6	10	15,428	104
>2	7	12	7,827	243
Laggers				
<2	3	2	7,893	63
>2	1	4	2,690	186
Females				
Low to moderate	1	0	2,066	48
Severe				
<2	8	5	9,538	136
>2	4	3	4,388	360

SOURCE: Muriel L. Newhouse and Geoffrey Berry, "Patterns of Mortality in Asbestos Factory Workers in London," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 57 (Table 5).

told the Commission, there is biologically no reason to doubt that the incidence of mesothelioma is dose-related.[302]

We recognize that the reported deaths from mesothelioma after what appear to have been brief (for gas mask workers) or low (for family contact and neighbourhood cases) exposures have caused considerable public anxiety concerning this disease. We cannot confirm the accuracy or otherwise of the proposition that there are cases of mesothelioma which have occurred from insignificant doses of asbestos. However, we think it unlikely. The doses to which the gas mask workers were exposed appear to have been quite intense, albeit brief, in duration;[303] pictorial evidence and subsequent experiments have indicated that household and neighbourhood exposures where mesothelioma resulted were in fact considerably higher than originally thought and approached, or in many cases were equivalent to, corresponding occupational exposures.[304]

Dr. Davis told the Commission that the notion that mesothelioma occurs at low doses first arose from the documented cases of mesothelioma among those who lived in the vicinity of the Cape crocidolite mines in South Africa and from the cases occurring among the wives of asbestos workers who merely brushed their husbands' overalls when they came home from work at night. Dr. Davis is of the opinion that the wrong conclusion has been drawn from this evidence.[305] He has seen pictures which demonstrate that in the past, crocidolite waste was used to surface roads in South Africa and that vehicles driving along these roads produced dust clouds likely as high as in asbestos mines or factories. Further, Dr. Davis is aware of experiments which have shown that exposing overalls to asbestos clouds of the sort that might have occurred years ago and then brushing them produced dust levels in the order of 200 f/cc.[306]

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[302] See RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), p. 81; and RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 66. Dr. Nicholson indicated that while the data on the dose-response relationship for mesothelioma are very scanty, those data that do exist are completely consistent with a linear dose-response relationship. See also, RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 15 to the same effect. And see Sheers and Coles, "Mesothelioma Risks in a Naval Dockyard," in which mesothelioma incidence rates were correlated both with duration of employment in exposed occupations and the relative severity of exposure of various shipyard jobs. There is also some suggestion of a relationship between the extent of the exposure and the site of a mesothelioma tumour. Dr. J.C. McDonald testified that in a U.K. study of all the cases of mesothelioma known to the Cape Asbestos Company, the heavily exposed individuals had a high proportion of peritoneal mesotheliomas; the victims who were lightly exposed, even to crocidolite, tended to have pleural mesotheliomas. See RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 102.

[303] Mr. Peto testified that the mesothelioma cases among the gas mask workers, almost without exception, had very high fibre content in their lungs, so that they were apparently exposed to very high doses over a short period. See RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 37.

[304] See RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 16; RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 92–94; RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), p. 106; and RCA Transcript, Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41, pp. 77–81.

While all of this evidence suggests that the incidence of mesothelioma is dose-related, none of it sustains the proposition that there is a threshold for this disease. We accept this fact and would therefore expect to see a few persons contracting mesothelioma after brief or low exposures. But as Mr. Peto observed in his testimony:

> To assume on the basis of anecdotes that people who drilled a hole in an asbestos roof when they were 20, who get mesotheliomas 50 years later due to that exposure, I think it's completely unreasonable. I think it's very unlikely that the effect of brief exposure is more than proportional, and the majority of cases seem to be occurring in people who have had fairly substantial exposure.[307]

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[305] RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 92–94.

[306] Ibid. See also, RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, pp. 91–92. Dr. J.C. McDonald also testified that the concentrations of asbestos fibres in the homes of asbestos workers can be very high because all the soft materials accumulate fibres which may then be shaken out. See also, RCA Exhibit II-57, Tab 8, in RCA Transcript, Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41: Ross Hunt, "Differences Between the Re-Aerosolability of Settled Airborne Chrysotile and Amphibole Dusts," extracts from reports issued to the Asbestosis Research Council, Cleckheaton, Yorkshire, BBA Group PLC, Industrial Health Unit, 26 May 1982. (Mimeographed.) Mr. Hunt described an experiment wherein membrane samples were fastened to the handles of sweeping brooms, and sweeping operations were carried out using mixtures of crocidolite and chrysotile dust. The dust counts obtained showed that crocidolite became airborne in respirable dimensions very readily, while chrysotile, although airborne, was present mainly in accreted clusters which were not respirable. When cotton overalls were deliberately contaminated with the two fibre types and then subjected to moderately violent patting with the hands, the crocidolite fibres became airborne in respirable fractions as opposed to the chrysotile which flocculated and presented itself as non-respirable fibre aggregates. A room 9 feet square was used to demonstrate the re-aerosolability of crocidolite. Contaminated clothing was shaken in the room and subsequently a broom was used to move the dust from one part to another. Counts of 6 f/cc down to 2 f/cc were obtained over a period of two weeks.

[307] RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 131–132.

And as Dr. Finkelstein commented:

> My feeling is that you may certainly develop a mesothelioma with a short exposure, but the risk is substantially less than the risk at higher exposures. . . .[308]

Of course, such cases that do occur tend to stand out, more, for example, than lung cancer, because of the asbestos-specific nature of the disease. On our assessment of the available data, we have no reason to disagree with the conclusion reached in the "Report of the Advisory Committee on Asbestos Cancers to the Director of the International Agency for Research on Cancer" in 1973 that insofar as mesothelioma is concerned, "There is no evidence of a risk to the general public at present."[309]

E.4 Age and Time Dependency of the Response

As we have observed earlier in this Report, there is a time interval between the initial exposure to asbestos and the clinical manifestation of the diseases it causes. This time interval is commonly known as latency, and diseases from inhaling asbestos, like diseases from many other agents that cause pneumoconiosis and cancer, are thought to have a very long latency period.[310] These latency periods are difficult to define or measure precisely, but on the basis of observed cases for asbestosis and asbestos-related cancers other than mesothelioma, they are rarely less than 10 years and often more than 20 years. Mesotheliomas appear to have the widest range of latency — again, they rarely occur less than 10 years from the time of first exposure to asbestos, but they can occur as many as 40 years or more from the onset of exposure.[311] In the 21 cases of mesothelioma reported by

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[308] RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 91.

[309] "Report of the Advisory Committee on Asbestos Cancers to the Director of the International Agency for Research on Cancer," in *Biological Effects of Asbestos*, p. 342. See also, RCA Transcript, Evidence of Dr. J. Corbett McDonald, 25 June 1981, Volume no. 13, p. 74. Dr. J.C. McDonald testified that the contribution of occupational exposure is the main factor affecting the increased rate of mesothelioma in males, and the absence of an increased rate in females implies that non-occupational exposure is probably not having any important effect in North America.

[310] See U.S., Department of Labor, Occupational Safety and Health Administration, "Identification, Classification and Regulation of Potential Occupational Carcinogens," 29 CFR Part 1990, 45 FR 5002–5296, 22 January 1980 [hereafter "The OSHA Cancer Policy"]. It is stated in "The OSHA Cancer Policy" that "The fourth distinctive characteristic of cancer, the long *latent periods* between exposure and effect, was abundantly documented in the Record. Many witnesses pointed out that there is a long latent period between the initial events in carcinogenesis and the clinical manifestations of the disease. . . ." (p. 5026.)

[311] RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 14–15; Alison D. McDonald, "Mesothelioma Registries in Identifying Asbestos Hazards," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 441; and Zielhuis, *Public Health Risks of Exposure to Asbestos*, pp. 78–80.

Dr. Finkelstein from Scarborough, the latency period ranged from 14 to 32 years; in all but 3 of these cases death occurred before age 65 and at least 5 deaths occurred while the individuals were in their 40s.[312] By contrast, all 5 cases of pleural mesothelioma among the household members of the Paterson, New Jersey, amosite asbestos workers occurred 30 or more years from the time of first exposure.[313] And of the 16 cases of mesothelioma among the Quebec chrysotile miners, 4 occurred 30 to 39 years from first exposure; 5 occurred 40 to 49 years from first exposure; and 6 occurred 50 or more years from first exposure.[314]

The obvious effect of latency is that the asbestos-related diseases we observe today are the result of exposures many years ago; correspondingly, whether the exposures of the past few years might lead to asbestos-related disease cannot be known with certainty for years to come.

Recently, a number of experts working in the field of cancer have reconsidered the traditional concept of latency and have viewed the time interval between first exposure and clinical manifestation of the disease from a new perspective. Cancer is a disease that occurs predominantly in the middle and older age groups,[315] and these experts believe that what has been called a latency period is simply a difference in the time between first exposure and the time when the disease most often occurs.[316]

Moreover, in the case of asbestos, the time considerations that apply to lung cancer appear to be quite different from those that apply to mesothelioma. Recent work, both by Mr. Julian Peto while he was at the University of Oxford and by Dr. William J. Nicholson at the Mount Sinai School of Medicine in New York, suggests that the death rates from mesothelioma appear to rise at an exponential rate from the time since first exposure.[317] Mr. Peto's work suggests that mesothelioma death rates rise at a rate between the third and fourth power of time since first exposure; and Dr. Nicholson's work suggests the fifth power of time. What both have demonstrated is that the incidence of mesothelioma rises rapidly the longer the time period since a person is first exposed to asbestos. As a result, the age at which a person is first exposed to asbestos becomes a very significant factor in determining the overall risk of contracting mesothelioma. Utilizing the data from Dr. Selikoff's study of North American insulation workers, Mr. Peto has been able to demonstrate that the risk of contracting mesothelioma is approximately 10 times higher for persons first exposed to asbestos when of school age as compared to persons first exposed when over age 40.[318] This is illustrated in Table 5.32. While the mesothelioma incidence rates appear to be independent of the age at which exposure first took place, the practical result is that the risk of contracting mesothelioma is greater the earlier in life one is first exposed. The magnitude of the risk will still depend on the amount and duration of exposure (and, it may be added, the type of fibre); and where that exposure is minimal, the risk,

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[312] Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," p. 14. This study confirms that 18 of the 21 mesothelioma deaths occurred below the age of 65. An earlier version of the same study presented at the Commission's hearings indicated that of the first 16 deaths from mesothelioma at the Scarborough plant, 5 of the victims were in their 40s at the time of death. See RCA Exhibit II-36, Tab 7, in RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 79: Murray M. Finkelstein, "Mesothelioma Deaths Among Johns-Manville Workers." (Table, Mimeographed.)

[313] RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, p. 32. See also, Anderson et al., "Asbestosis Among Household Contacts of Asbestos Factory Workers."

[314] A.D. McDonald, "Malignant Mesothelioma in Quebec," p. 676.

[315] John Cairns, *Cancer: Science and Society* (San Francisco: W.H. Freeman and Company, 1978), chap. 2, pp. 5-14. See also, Peto, Seidman, and Selikoff, "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment," pp. 131-132.

[316] Mr. Peto in fact testified that latency as the term is normally used is not a helpful concept. See RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 66-70.

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[317] See Julian Peto, Brian E. Henderson, and Malcolm C. Pike, "Trends in Mesothelioma Incidence in the United States and the Forecast Epidemic Due to Asbestos Exposure During World War II," in *Banbury Report 9: Quantification of Occupational Cancer*, pp. 51–69; Peto, Seidman, and Selikoff, "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment," pp. 124–135; Julian Peto, "Dose and Time Relationships for Lung Cancer and Mesothelioma in Relation to Smoking and Asbestos Exposure," in *Zur Beurteilung der Krebsgefahren durch Asbest* [Proceedings of the Bundesgesundheitsamt Asbestos Symposium], Berlin: February 1982, bga Schriften MMV Medizin Verlag München, in press, 1983; William J. Nicholson et al., "Cancer from Occupational Asbestos Exposure: Projections 1980–2000," in *Banbury Report 9: Quantification of Occupational Cancer*, pp. 87–108; and RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 36–37, 40–41.

[318] See RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 10–12; and Julian Peto, "An Alternative Approach for the Risk Assessment of Asbestos in Schools," report to the U.S. Environmental Protection Agency, 6 April 1981. (Mimeographed.)

Table 5.32

Predicted Numbers of Mesotheliomas That Would Occur Among a Given Number of Men in Each Quinquennium Following First Exposure to Asbestos at a Fixed Level, by Age at First Exposure

[Incidence Assumed to Increase as (Time Since First Exposure)$^{3.5}$]

	Age First Exposed							
	16-20	21-25	26-30	31-35	36-40	41-45	46-50	51-55
	Median Age First Exposed							
	18½	23½	28½	33½	38½	43½	48½	53½
Age at Diagnosis	**Predicted Numbers of Mesotheliomas**							
25-29	0.04							
30-34	0.17	0.04						
35-39	0.48	0.17	0.04					
40-44	1.07	0.47	0.16	0.04				
45-49	2.02	1.05	0.47	0.16	0.03			
50-54	*3.40**	1.97	1.02	0.46	0.16	0.03		
55-59	5.19	*3.24*	1.87	0.97	0.43	0.15	0.03	
60-64	7.22	4.77	*2.98*	1.72	0.90	0.40	0.14	0.03
65-69	9.12	6.31	4.17	*2.60*	1.51	0.79	0.36	0.13
70-74	10.32	7.40	5.12	3.39	*2.12*	1.23	0.65	0.30
75-79	10.15	7.50	5.39	3.73	2.47	*1.55*	0.91	0.49
Total below age 80	49.18	32.92	21.22	13.07	7.62	4.15	2.09	0.95

Note: *Figures corresponding to cases occurring in 1974-1978 in men first exposed in 1942 are italicized.

SOURCE: Julian Peto, Brian E. Henderson, and Malcolm C. Pike, "Trends in Mesothelioma Incidence in the United States and the Forecast Epidemic Due to Asbestos Exposure During World War II," in *Banbury Report 9: Quantification of Occupational Cancer*, eds. Richard Peto and Marvin Schneiderman ([Cold Spring Harbor, New York]: Cold Spring Harbor Laboratory, 1981), Table 1, p. 53.

albeit greater for exposures earlier rather than later in life, will also be minimal.[319] The time-dependence of mesothelioma appears to provide a reasonable explanation for the observed incidence of the disease in industrial cohorts and also appears to accord with current models of carcinogenesis. Accordingly, we use a model that incorporates the time-dependence (and age-independence) of mesothelioma in our risk assessment found in Chapter 7.

The disease rate of lung cancer among persons exposed to asbestos appears to be quite unlike that of mesothelioma. Rather than being time-dependent, lung cancer rates appear to be age-dependent.[320] The majority of lung cancer deaths, both in smokers and non-smokers, occur after age 50 and over half occur after age 60, irrespective of the time of first exposure.[321] This suggests that the risk of contracting lung cancer is much greater in older groups than in younger groups. Asbestos exposure appears to have the effect of multiplying the risk of lung cancer which exists apart from that exposure; and the risk of lung cancer contributed to by asbestos exposure appears to be virtually independent of the age when that exposure took place and will be simply proportional to cumulative dose.[322] Accordingly, even if a person is first exposed to asbestos quite late in life, the effect of that exposure in terms of the risk of contracting lung cancer may be quite substantial because it will have the effect of multiplying the already elevated lung cancer risk that pertains to older age groups. By contrast, the risk of contracting lung cancer at age 20 or age 40 from asbestos exposure is quite negligible because the young have a minimal risk of the disease apart from asbestos exposure.[323] As the model is in accordance with accepted principles of carcinogenesis and is consistent with the epidemiological data, it forms the basis for our risk assessment for lung cancer in Chapter 7.

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[319] In his paper, "An Alternative Approach for the Risk Assessment of Asbestos in Schools," Mr. Peto calculated the mesothelioma risk up to age 80 due to exposure at 1 f/cc for 6 years from age 12, and from exposure to 1 f/cc for 10 years from age 30. On his calculation, the earlier exposure would produce 329.1 total deaths per 100,000 and the later exposure, 107.2 deaths per 100,000. Since asbestos levels in schools are always 1/100 and usually very much less than 1/1,000 of 1 f/cc, the lifelong risk would accordingly be at least 100 and probably more than 1,000 times lower than this calculation and thus is likely to be less than 1 in 100,000, which seems to be negligible. See Table 4, p. 13. In "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment," Peto, Seidman, and Selikoff clearly stated that their model does not mean that the risk is unrelated to fibre type and intensity of exposure. Their calculations were based upon the mortality experience of the North American insulation workers exposed to amosite. The authors further indicated that if the contribution to subsequent mortality due to each inhaled asbestos fibre is proportional to the cube of time since inhalation, the death rate following brief exposure would rise as the cube of time since first exposure; the rate due to continuous exposure would rise as the fourth power of time; and the effect of intermediate duration would be well approximated by an exponent of time between 3 and 4.

[320] See Peto, "An Alternative Approach for the Risk Assessment of Asbestos in Schools," p. 1; and RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 16–22.

[321] See RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 65.

[322] See RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 27–28. The dose, of course, still remains important with respect to lung cancer as well as mesothelioma. Dose affects the relative risk. The higher the dose, the higher the relative risk. But the dose itself does not affect the latency period. Thus, it is incorrect to suggest that if one reduces exposure, one increases the latency period. A reduction in exposure simply reduces the relative risk of contracting the disease. See ibid., pp. 30–34; and RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 66.

F. The Effect of Smoking

It is now a well-accepted fact that cigarette smoking plays a critical role in the incidence of lung cancer.[324] Smoking has been a common habit among working populations including those individual cohorts of asbestos workers that have been subjected to epidemiological study. It therefore becomes important to consider the extent to which the incidence of lung cancer among asbestos workers is attributable to their smoking habits rather than to their asbestos exposure. Beyond that, it is relevant to explore the extent to which smoking affects the risk of contracting the other asbestos-related diseases.

The seminal study on the relationship between smoking and asbestos disease is that of Hammond, Selikoff, and Seidman.[325] These authors obtained complete smoking histories for 8,220 men out of a cohort of 12,051 North American asbestos insulation workers who had 20 or more years of employment. As a control population, the authors used the 73,763 men in the American Cancer Society's prospective cancer prevention study.[326] The authors found that asbestos workers who did not smoke had approximately a 5 times greater risk of dying of lung cancer than the non-smoking control population. Similarly, asbestos workers who did smoke had about a 5 times greater risk of dying of lung cancer than the smoking control population. Smokers were at 11 times greater risk of lung cancer than non-smokers; and the risk for smoking asbestos workers was elevated approximately 55-fold over the non-smoking control population.[327] Table 5.33, which was referred to by several witnesses during our hearings, illustrates these differences.

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[323] RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 65; and RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 22.

[324] See, for example, Richard Doll and Richard Peto, "Cigarette Smoking and Bronchial Carcinoma: Dose and Time Relationships Among Regular Smokers and Lifelong Non-Smokers," *Journal of Epidemiology and Community Health* 32 (1978): 303–313. See also, Richard Doll and Richard Peto, "The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today," *Journal of the National Cancer Institute* 66:6 (June 1981): 1191–1308. The authors have stated that:

> No single measure is known that would have as great an impact on the number of deaths attributable to cancer as a reduction in the use of tobacco or a change to the use of tobacco in a less dangerous way. The principal impact would be on the incidence of cancer of the lung, which by late middle age is more than ten times greater in regular cigarette smokers than in lifelong non-smokers, but a material effect would also be produced on the incidence of cancers of the mouth, pharynx, larynx, esophagus, bladder, probably the pancreas, and perhaps the kidney. (p. 1220.)

[325] E. Cuyler Hammond, Irving J. Selikoff, and Herbert Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 473–490.

These rates are applicable to the particular cohort of insulation workers that was studied, and we would not be justified in applying them to other populations with different smoking habits, different levels of asbestos exposure, and different lung cancer risks.[328] Nonetheless, in our judgement, the Hammond study does indicate five relevant findings of more general application concerning asbestos exposure, smoking, and lung cancer. They are as follows:

(i) The relative risk of contracting lung cancer from asbestos is approximately the same for both smokers and for non-smokers. In other words, asbestos multiplies by the same amount the risk of contracting lung cancer for smokers and non-smokers alike.[329] In turn, this evidences the fact that asbestos on its own, in the absence of smoking, is capable of inducing lung cancer, a finding that has been amply confirmed both on the basis of experimental models and other cohort studies.[330]

(ii) While there is a risk of lung cancer for non-smoking asbestos workers, the great preponderance of lung cancers, in absolute terms, occurs in those asbestos workers who have smoked.[331] In the Hammond study, of the 276 asbestos workers who, according to death certificate information, died of lung cancer and whose smoking habits were known, only 4 never smoked regularly and only 4 others smoked only a pipe or cigar. By comparison, there were 152 workers who were current smokers of 20 or more cigarettes a day at the time of their death.[332] Accordingly, while asbestos

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[326] Ibid., pp. 473–475.

[327] Ibid., pp. 486–487.

[328] See RCA Transcript, Evidence of Mr. Geoffrey Berry, 19 June 1981, Volume no. 11, p. 21.

[329] See Peto, "Dose and Time Relationships for Lung Cancer and Mesothelioma in Relation to Smoking and Asbestos Exposure," p. 2.

[330] RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), pp. 19–20; and 22 July 1981, Volume no. 21(A), p. 76. See also, J. Corbett McDonald, "Asbestos and Lung Cancer: Has the Case Been Proven?" *Chest* 78:2 (August 1980, Supplement): 374–376; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 64.

[331] RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), pp. 19–20.

[332] Hammond, Selikoff, and Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," Table 7A, p. 486.

Table 5.33

Age-Standardized Lung Cancer Death Rates* for Cigarette Smoking and/or Occupational Exposure to Asbestos Dust Compared with No Smoking and No Occupational Exposure to Asbestos Dust

Group	Exposure to Asbestos?	History Cigarette Smoking?	Death Rate	Mortality Difference	Mortality Ratio
Control	No	No	11.3	0.0	1.00
Asbestos workers	Yes	No	58.4	+47.1	5.17
Control	No	Yes	122.6	+111.3	10.85
Asbestos workers	Yes	Yes	601.6	+590.3	53.24

Note: *Rate per 100,000 man-years standardized for age on the distribution of the man-years of all the asbestos workers. Number of lung cancer deaths based on death certificate information.

SOURCE: E. Cuyler Hammond, Irving J. Selikoff, and Herbert Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 487 (Table 8).

exposure does increase the risk of contracting lung cancer in smokers and non-smokers alike, because the absolute risk of lung cancer is so small in non-smokers, the contribution of asbestos in absolute numbers is also small.[333]

(iii) In combination, asbestos exposure and smoking act synergistically in causing lung cancer. In the actual study by Dr. Hammond, the numbers are consistent with a multiplier effect between asbestos and smoking (with death rates of 11.3, 58.4, 122.6, and 601.6 per 100,000 men for non-exposed non-smokers, asbestos-exposed non-smokers, non-exposed smokers, and asbestos-exposed smokers respectively).[334] The significance of the interaction between asbestos and smoking becomes even clearer by expressing the results of the Hammond study in a different fashion. Of the 1,946 observed deaths in the cohort of insulation workers studied, 450 deaths, according to the best available evidence, were caused by lung cancer. Dr. Selikoff, in a separate report, subsequently considered how many lung cancer deaths would have occurred in the cohort absent smoking and/or asbestos.[335] According to his calculations, if there had been asbestos exposure and no cigarette smoking there would have been 44 lung cancer deaths; if there had been no asbestos exposure but cigarette smoking there would have been 94 deaths; and if there had been neither cigarette smoking nor asbestos exposure there would have been only 9 lung cancer deaths.

Mr. Berry's studies in England have similarly indicated that asbestos and smoking combine multiplicatively to produce lung cancer,[336] and the data from Dr. J.C. McDonald's Quebec chrysotile mining study are compatible with a multiplicative model.[337] Whether an exact multiplier effect will hold for every individual cohort is not overly important. What is important is that there is a very powerful interaction between cigarette smoking and asbestos which produces an effect that is certainly greater than the sum of their individual contributions. This conclusion, drawn from various cohort studies, is also supported by experimental evidence which suggests that asbestos particle clearance in smokers is considerably lower than in non-smokers.[338] This suggests that smoking may impair the clearance of asbestos fibres from the lung. Dr. Kotin testified that chronic smoking over a period of time will initiate a process that results in the death of the ciliated cells, thereby reducing the effectiveness of the important clearance mechanisms in the lung.[339] He also suggested that cigarette smoke would interfere with the other main defence against asbestos fibres, the macrophages. As cigarette smoke is also a material foreign to the body, the same macrophages used to clear asbestos fibres will be needed to clear cigarette smoke.[340] Still, the exact manner in which asbestos and cigarette smoke combine in synergistic fashion to produce lung cancer remains uncertain.[341]

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[333] As Mr. Peto stated in "Dose and Time Relationships for Lung Cancer and Mesothelioma in Relation to Smoking and Asbestos Exposure": "For lung cancer, the relative risk (ratio of incidence to incidence in unexposed individuals of the same age and smoking habits) is more or less independent of age and cigarette smoking, but the absolute risk in the general population is strongly related to both age and cigarette smoking." (p. 2.) See also, RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, p. 73; and J.C. McDonald, "Asbestos-Related Disease: An Epidemiological Review," p. 590.

[334] See Table 5.33 in this chapter.

[335] Irving J. Selikoff, "Two Comments on Smoking and the Workplace," *American Journal of Public Health* 71:1 (January 1981): 92.

[336] See RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, pp. 109–110. See also, Geoffrey Berry, Muriel L. Newhouse, and Mary Turok, "Combined Effect of Asbestos Exposure and Smoking on Mortality from Lung Cancer in Factory Workers," *The Lancet* 2:775 (2 September 1972): 476–479. The authors studied the smoking habits of over 1,300 male and 480 female factory workers and their mortality from lung cancer over a 10-year period. No significant excess was found in workers, whether smokers or non-smokers, with low to moderate asbestos exposure, but among workers who smoked and who were severely exposed the excess was highly significant. The analysis, particularly of the data relating to women where there was a high proportion of non-smokers, supports the multiplicative hypothesis for the action of asbestos and tobacco. See also, Newhouse and Berry, "Patterns of Mortality in Asbestos Factory Workers in London," p. 59.

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[337] RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 63–64. In fact, Dr. J.C. McDonald suggested that, on balance, his data are more compatible with an additive model than with a multiplicative one. The following table of relative risks illustrates this:

	Asbestos Exposure		
	Little	Moderate	Heavy
Non-smokers	1	2.0	6.9
Moderate smokers	6.3	7.5	12.8
Heavy smokers	11.8	13.3	25.0

SOURCE: J. Corbett McDonald, "Asbestos-Related Disease: An Epidemiological Review," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), p. 590.

In the end, Dr. J.C. McDonald concluded that one can certainly say that there is a very important interaction between smoking and asbestos and it could be anything between additive and multiplicative.

[338] Richard A. Lemen et al., *Workplace Exposure to Asbestos: Review and Recommendations*, prepared by the NIOSH/OSHA Asbestos Work Group, April 1980, DHHS (NIOSH) Publication no. 81–103 (Washington, D.C.: U.S. Department of Health and Human Services and U.S. Department of Labor, November 1980), pp. 27–28. See also, reference made therein to a study by D. Cohen, S.F. Arai, and J.D. Brain, "Smoking Impairs Long-Term Dust Clearance from the Lung," *Science* 204 (4 May 1979): 514–517.

[339] See RCA Transcript, Evidence of Dr. Paul Kotin, 22 July 1981, Volume no. 21(A), pp. 62–68. Thus, the cigarette smoker would have a higher retention of asbestos fibres than would the non-smoker. If an individual is a cigarette smoker, his ability to clear asbestos fibres from his lungs will be impaired and he will accordingly have a greater effective dose of asbestos in his lungs.

[340] Ibid., p. 73.

[341] Ibid., p. 75. See also, John E. Craighead and Brooke T. Mossman, "The Pathogenesis of Asbestos-Associated Diseases," *The New England Journal of Medicine* 306:24 (17 June 1982): 1451.

(iv) It is evident that of the two variables, cigarette smoking and asbestos exposure, the former appears to have the more powerful effect on the incidence of lung cancer.[342] For example, in the Hammond study, smoking increased the incidence of lung cancer by a factor of 11; asbestos, by a factor of 5.[343] While we have no reason to doubt this observation at the present time, a word of caution is in order. Dr. Enterline testified that for a long period of time Dr. Selikoff did not observe any deaths among his non-smoking insulation workers, although he now clearly does. Dr. Enterline compared this to a similar pattern he has observed among uranium miners where there is also a synergistic effect between radiation and smoking. According to Dr. Enterline, it now appears that non-smoking uranium miners are beginning to experience a very high incidence of lung cancer, and he speculates that what we may be observing in uranium miners is the disease incidence further downstream. He has suggested that we may also witness a downstream incidence of lung cancer among non-smoking asbestos workers.[344] At the present time that suggestion remains in the realm of speculation.

(v) To the extent that asbestos-exposed persons stop smoking, their risk of death from lung cancer, like that of non-asbestos exposed persons who stop smoking, decreases quite considerably.[345] Again, utilizing the North American insulation workers data, the risk of lung cancer mortality for those workers who had previously smoked but stopped in 1967 was approximately one-third that of their workmates who continued to smoke over the next 10-year period.[346]

Smoking appears to have a different relationship to asbestosis than it does to lung cancer. Smoking can clearly initiate lung cancer; it does not initiate the fibrotic process that leads to asbestosis. Further, there are some studies such as that of Dr. Weill which evidence no relationship at all between smoking and the progression of the fibrotic process.[347] However, the weight of the available data indicates that cigarette smoking may affect the progression of the fibrotic process, both in terms of whether clinical asbestosis becomes manifest and of its subsequent rate of progression. The Hammond study found that asbestosis death rates were nearly 3 times higher for heavy smokers than for non-smokers, prompting the authors of the study to conclude that cigarette smoking greatly increases the risk of an asbestos worker dying from asbestosis or from fibrosis in combination with emphysema resulting from smoking.[348] Mr. Berry considered the effect of smoking on the incidence of clinical asbestosis at Rochdale. For men first employed after 1950 with at least 10 years' employment by 1966, very little evidence of asbestosis was found other than in smokers.[349] (See Table 5.34.) On the basis of the data from Rochdale and similar data from the Royal Navy dockyard workers studied by Rossiter, Berry concluded that smokers had a higher risk of developing clinical asbestosis during their lifetime than non-smokers, and his results indicated that for a non-smoker with less than 100 f/cc-yrs exposure, the risk of developing the clinical manifestation of asbestosis was minimal.[350]

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[342] RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, p. 84; and RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, pp. 53–55.

[343] That smoking is a more powerful variable than asbestos exposure is also illustrated in the study by Dr. J.C. McDonald et al. of the Quebec chrysotile miners. See note 337, *supra*.

[344] RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 88–90.

[345] Dr. Nicholson testified that if one looks at the mortality rate for lung cancer in individuals who stop smoking cigarettes, one finds that it can fall dramatically over a 10-year period to perhaps a quarter or even less than that which it would have been had one continued to smoke, whatever one's cigarette habit had previously been. After 5 years of cessation of cigarette smoking, the risk of death from lung cancer decreases to perhaps one-half or one-third what it would have been had one continued to smoke cigarettes. RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, pp. 55–56.

[346] Irving J. Selikoff, "Asbestos-Associated Disease," in *Public Health and Preventive Medicine*, 11th ed., ed. John M. Last (New York: Appleton-Century-Crofts, 1980), chap. 13, p. 579.

The data pertaining to the possible association between asbestos and smoking in relation to laryngeal cancer and cancer of the gastrointestinal tract are scanty. It does, however, appear that most of those in whom carcinoma of the larynx is associated with asbestos exposure have been smokers.[351] Dr. Selikoff's work suggests that the increase in cancer of the esophagus among asbestos workers occurs only in smokers. No such association was demonstrated, however, for cancer of the stomach, colon, or rectum.[352]

In striking contrast to lung cancer, there is no evidence to demonstrate any relationship whatsoever between cigarette smoking and mesothelioma.

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[347] RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, p. 25. See also, Weill et al., "Lung Function Consequences of Dust Exposure in Asbestos Cement Manufacturing Plants," p. 96.

[348] Hammond, Selikoff, and Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," pp. 484–485.

[349] RCA Transcript, Evidence of Mr. Geoffrey Berry, 19 June 1981, Volume no. 11, pp. 23–26; and Berry et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory," p. 104.

[350] Charles E. Rossiter and Geoffrey Berry, "The Interaction of Asbestos Exposure and Smoking on Respiratory Health," *Bull. europ. Physiopath. resp.* 14 (1978): 202; and Berry et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory."

[351] RCA Transcript, Evidence of Dr. Alexander C. Ritchie, 14 July 1982, Volume no. 49, pp. 20–21.

[352] Ibid. See also, Selikoff, "Asbestos-Associated Disease," p. 577.

Table 5.34

The Association Between Smoking, Age, and Signs of Asbestosis

Employment Group and Smoking Habit	Age Group	Numbers of Men				
		Total	Crepitations	Possible Asbestosis	Certified Asbestosis	Small Radiological Opacities*
First employed before 1951						
Never smoked and 1-4 cigarettes per day	-54	11	0	0	1	0
	55+	18	7	6	2	6
Other smokers and ex-smokers	-54	37	7	5	2	6
	55+	115	42	34	19	47
First employed after 1950						
Never smoked and 1-4 cigarettes per day	-44	26	0	0	0	1
	45-54	17	0	0	0	0
	55+	12	0	0	0	1
Other smokers and ex-smokers	-44	32	0	0	0	1
	45-54	51	8	5	4	10
	55+	57	18	8	6	15

Note: * Profusion 1/10 or more.

SOURCE: Geoffrey Berry et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory," *British Journal of Industrial Medicine* 36 (May 1979): 104 (Table 7).

Indeed, all available data and all expert opinions support the conclusion that the incidence of mesothelioma is entirely independent of smoking.[353]

G. What are the Health Effects of Peak Exposures?

High exposures of brief or intermittent duration can and often do occur in some parts of the asbestos industry, particularly in construction, maintenance, demolition, and repair work.[354] This has led us to inquire whether or not these more intense exposures of asbestos — or short, sharp bursts as we referred to them during our hearings — may carry with them a disproportionate health risk. Is a construction worker who, for example, has a total asbestos exposure of 100 f/cc-yrs but who accumulated this exposure in short yet high doses at greater risk than a factory worker who accumulated the same total exposure in continuous and lower doses (and generally over a longer period of time)?

Unfortunately, the epidemiological studies of asbestos workers are of qualified assistance in answering this question. Even for those cohorts where exposure measurements have been available, they have not differentiated between short, intense exposures and more continuous but lower exposures. Consequently, the epidemiologist has been left with only a single average or cumulative figure. Despite the lack of direct data, we nonetheless have a concern on the basis of scattered evidence and on the basis of what is known about the clearance mechanisms in the lung that peak exposures may well produce a disproportionate amount of disease. That concern was raised by a number of the witnesses who appeared before us. For example, Drs. Becklake, Enterline, Kotin, and Weill all suggested that excessive amounts of asbestos dust inhaled in any one period of time may overload the lung's clearance mechanisms, thereby increasing the amount of asbestos fibres that are retained within the body compared with the retention that takes place at continuous but lower exposures.[355]

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[353] RCA Transcript, Evidence of Mr. Geoffrey Berry, 19 June 1981, Volume no. 11, p. 45; and RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 70. See also, Peto, "Dose and Time Relationships for Lung Cancer and Mesothelioma in Relation to Smoking and Asbestos Exposure," p. 2; and Hammond, Selikoff, and Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," p. 487.

[354] See, for example, RCA Transcript, Evidence of Dr. William J. Nicholson, 30 June 1981, Volume no. 15, p. 90.

Moreover, there does appear to be some epidemiological evidence, indirect in nature, to support the notion that short, sharp bursts may carry a disproportionate health risk. In Dr. Enterline's earlier study of the Manville factory retirees, excess mortality for all causes of death among maintenance-service workers was 24.3%, whereas for production workers it was only 10.7%; for lung cancer, excess mortality for maintenance-service workers was 328.6%, while for production workers it was 73.7%.[356] Dr. Enterline suggested that the intermittency and possible severity of maintenance workers' exposure may well account for this difference.[357] This observation must, as Dr. Enterline pointed out, be treated with caution because of the possible confounding effect of fibre type — only 26% of the production workers in Dr. Enterline's cohort were exposed to amosite and/or crocidolite, compared to 71% of maintenance workers.[358] Support for Dr. Enterline's suggestion may be found in the results of Dr. Weill's study of the asbestos-cement pipe factory at New Orleans where the maintenance workers exposed intermittently to crocidolite had a substantially higher risk of lung cancer than those in the plant steadily exposed to crocidolite.[359]

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[355] RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, pp. 46–47; RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 41–42; RCA Transcript, Evidence of Dr. Paul Kotin, 23 July 1981, Volume no. 21(B), p. 36; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 62–63. Dr. Davis testified that he attempted to examine the effects of intermittent, high asbestos exposures (peak dose levels) in his animal experiments. The findings of his study appeared to refute the idea that short periods at very high exposure levels to asbestos result in a much higher lung burden of retained dust than might be expected from the greater density of the "peak dust clouds." However, in the experimental situation, it was impossible to examine the effects of the realistic differential between ordinary and peak exposures that one sees in the human situation because it is necessary that the ordinary exposure of animals be relatively elevated. In his experiments, Dr. Davis had only a fivefold difference between the peak and the ordinary exposure of his animals, whereas in factories the overall dust levels are sufficiently low that a peak dose caused by, for example, a temporary machine defect could easily be 100 times higher than the ordinary level. See John M.G. Davis et al., "The Effects of Intermittent High Asbestos Exposure (Peak Dose Levels) on the Lungs of Rats," *British Journal of Experimental Pathology* 61 (1980): 272–280; and RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 103–104.

[356] Enterline, DeCoufle, and Henderson, "Mortality in Relation to Occupational Exposure in the Asbestos Industry," pp. 901–902.

[357] Ibid., p. 900. See also, RCA Transcript, Evidence of Dr. Philip E. Enterline, 11 June 1981, Volume no. 8, pp. 41–42.

[358] Enterline, DeCoufle, and Henderson, "Mortality in Relation to Occupational Exposure in the Asbestos Industry," p. 900.

A further piece of evidence that also lends support to the observation that peak exposures have particularly adverse health effects is the experience of the North American insulators which has been studied by Dr. Selikoff and his colleagues since the early 1960s. As we have already stated, these insulators were, and are, members of the International Association of Heat and Frost Insulators and Asbestos Workers, primarily employed in the building trades. Here too, the extensive use of amosite may well be a contributing factor to the disease incidence of workers. Moreover, as there is an absence of actual exposure information, there are no data to assess the effects of different exposures.[360] Yet given the nature of the trades in which these workers were engaged, it is not unreasonable to expect, and indeed Dr. Selikoff indicated, that they often experienced short, intense exposures.[361] We cite in Chapter 9 of this Report evidence as to how high these exposures can be — for example, 20 to 100 f/cc in spraying insulation on buildings.[362] The health effect of such peak exposures does offer one possible explanation of the very high cancer incidence among the insulators.

We recognize that this limited evidence by no means firmly establishes the adverse health effects of short, sharp bursts. Indeed, it may be argued that the excess disease incidence observed in maintenance, repair, and insulation workers in some cohorts is due not to intermittent, high exposures but to the fact that these jobs are by their nature dustier and the cumulative exposure of workers in these jobs is simply greater; or it may be due, as we have indicated, to their exposure to crocidolite and amosite. We have no way of knowing what the true situation is. Nor does the available evidence tell us anything about how high one might expect these peak exposures should be in order for them to have a differential impact on health. With the limitations of the evidence in mind, we nonetheless conclude that short, sharp bursts of exposure do present a special health risk to workers, one that our regulatory system ought to have regard to, in the control of asbestos exposures both in fixed and non-fixed workplaces.

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[359] Weill, Hughes, and Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," pp. 352-353. The SMR for lung cancer among those workers with intermittent exposure to crocidolite in the pipe plant was 304, and the SMR for lung cancer among those workers with steady employment in the pipe plant with crocidolite exposure was only 155. RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 62-63.

[360] On the basis of direct measurements during recent years and data from other studies using similar processes, historical levels to which the insulators were exposed have been estimated at between 10 and 15 f/cc for commercial and industrial construction and between 15 and 20 f/cc for marine construction. See Nicholson, "Criteria Document for Swedish Occupational Standards: Asbestos and Inorganic Fibers," pp. 39-41. In their most recent published article, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," Selikoff, Hammond, and Seidman have stated that ". . . analysis of available data, including reconstruction of work situations and extrapolation to the past of observations made more recently, suggest that insulation workers would have been exposed to dust levels of 4-12 [f/cc]. . . ." (p. 92.)

[361] Selikoff, Hammond, and Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," p. 93; and Nicholson, "Criteria Document for Swedish Occupational Standards: Asbestos and Inorganic Fibers," p. 39. Dr. Nicholson suggested that the exposures could be extremely high and could range, for example, for 2- to 5-minute concentrations of asbestos, from 50 to 100 f/cc.

[362] See Chapter 9, Section B.1 of this Report.

H. Is There a Special Health Risk to Children from Asbestos Exposure?

One of the concerns that prompted the appointment of this Commission was the possible health hazard to children from exposure to asbestos in Ontario schools. Whether such a hazard in truth exists will of necessity very much depend upon the level and extent of exposures in the school system. This subject we pursue in detail in Chapters 9 and 10. Suffice it to say here that, in general, asbestos exposure levels in the Ontario school system are very low.

In this chapter we pursue a different question and that is whether children are more susceptible than adults to the health effects of asbestos exposure. If such is the case, then whatever are the measured levels of exposure in schools and whatever is the relevant dose-response relationship, we would, out of prudence, have to take particular account of this susceptibility in formulating our recommendations.

From our review of the medical evidence and from our questioning of the various witnesses who appeared before us, we could find no substantive support for the proposition that the lungs of young children are in and of themselves more susceptible than those of adults to asbestos-related diseases.[363] Indeed, there is evidence from animal experiments that might well suggest a contrary conclusion.[364] Despite this lack of evidence, we cannot exclude the biological possibility that children have less immunity to asbestos-related diseases than adults. This possibility arises because the cellular turnover rates in children are generally higher than those in adults. In a growing organ this may well mean much higher rates of mutational changes of the type thought to initiate the development of malignant tumours.[365] We note that Dr. Acheson, in his oral testimony, suggested we would do well to take into account the possibility that children might be more susceptible than adults in an immunological sense. Indeed, he suggested that there may be a general susceptibility of both young and aged to carcinogenic substances.[366]

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[363] RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, p. 111; RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 122; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, p. 64.

[364] RCA Transcript, Evidence of Dr. Margaret R. Becklake, 21 July 1981, Volume no. 20, pp. 66–67.

Indirect support for this hypothesis may perhaps be found in the work of Wasserman et al. who examined some 30 cases of mesotheliomas reported in the literature in which the tumour developed in infancy or childhood.[367] While such cases are admittedly rare and their association with asbestos-exposed parents by no means certain, Wasserman found two conspicuous characteristics of these mesotheliomas: there was a very high degree of malignancy, the majority dying less than a year from the onset of symptoms, and the latency period from first possible exposure was very short, the maximum being 14 years.[368] The rapidity of the process at least suggests that when children do contract mesothelioma, their immunity to the disease is somewhat less than that observed in adults.

Quite apart from these rare cases of childhood mesothelioma, there are a small number of clearly documented cases of mesothelioma occurring in adults which have been attributed to exposure to asbestos in childhood (invariably the exposure has been traced either to the home or to the neighbourhood of asbestos factories or to waste disposal sites). For example, Dr. J.C. McDonald testified that as a result of his own inquiries into the incidence of mesothelioma in North America based on cases ascertained through pathologists, he found 5 mesothelioma deaths in persons whose fathers were asbestos workers. At least 3 of the workers were employed in the Quebec mines. Death in these 5 persons occurred at ages 31, 32, 41, 42, and 51, thus tending to confirm that exposure to asbestos took place in childhood.[369]

These cases of mesothelioma attributable to childhood exposure do not of themselves indicate any greater susceptibility to asbestos on the part of children, but they do serve to emphasize the importance of the age of first exposure to asbestos. As we have discussed earlier in this chapter, the incidence of mesothelioma appears to rise at a rate between the third and fourth power of time since first exposure. And while this incidence rate is of itself independent of the age at which exposure first took place and in that sense is inconsistent with the notion that there is an age-related susceptibility to mesothelial tumours, the result is that exposures earlier in life carry with them a greater risk than exposures later in life.[370] This is attributable not to any greater biological susceptibility of the young but to the time-dependence of mesothelioma. Even if we set aside this notion, the relatively long latency period (often in excess of 30 years) that seems characteristic of the mesotheliomas occurring among those exposed to asbestos outside the workplace leads to the common sense conclusion that the earlier in life the exposure, the more likely that mesothelioma will occur during a normal lifespan.

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[365] RCA Transcript, Evidence of Mr. Julian Peto, 30 July 1981, Volume no. 25(B), p. 125; and RCA Transcript, Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41, pp. 89–91.

[366] RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 26. In its written submission to this Commission, the American Industrial Hygiene Association, Ontario Local Section, suggested that there is some evidence that a child's lungs may preferentially retain long, thin fibres. See American Industrial Hygiene Association, Ontario Local Section, Written submission to the Royal Commission on Asbestos, #11, 12 January 1981, p. 7.

[367] M. Wasserman et al., "Mesothelioma in Children," in *Biological Effects of Mineral Fibres*, vol. 1, pp. 253–257.

[368] Ibid., p. 254.

[369] RCA Transcript, Evidence of Dr. J. Corbett McDonald, 24 June 1981, Volume no. 12, pp. 109–110.

There is a further and perhaps a related consideration that deserves mention. There is the possibility that the residence time of asbestos fibres in the lung is an important determinant of the adverse health effects of asbestos. Several witnesses who appeared before us suggested that earlier exposures may be more important in causing disease than later exposures because the longer the fibre might remain in the body, the greater the opportunity to induce adverse effects.[371] Mr. Berry has gone so far as to construct a number of sophisticated models of dose-response that take this factor into account.[372] The potential importance of the residence time of fibres in the lung seems to us plausible, although admittedly at the present time its actual significance is still largely a matter of hypothesis and speculation.

Taken together these various considerations might suggest a slightly higher risk of mesothelioma for children than for adults. But, at the same time, we emphasize that there is no evidence to suggest that the exposure levels to which children have been subjected in asbestos-containing schools or elsewhere, any more than the exposure levels to which others in the general public have been subjected, have posed a health risk. Children have been exposed to asbestos in schools in North America for at least 30 years. If the hypothesis that mesothelioma is time-dependent is correct, and if school asbestos exposure were a health risk, we would have expected a number of non-occupational cases of mesothelioma to develop (apart from cases arising from known domestic or neighbourhood exposures). The absence of such cases is strongly indicative of the fact that school exposures are so low that they are not a health problem. Indeed, based on the incidence of mesothelioma among the North American insulators, Mr. Julian Peto has calculated that the cumulative risk of mesothelioma up to age 80 for children exposed to asbestos in schools for 6 years from age 12 onwards is likely to be less than one in 100,000; in other words, negligible.[373]

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[370] RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 122; and RCA Transcript, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), p. 57.

[371] See RCA Transcript, Evidence of Mr. Geoffrey Berry, 18 June 1981, Volume no. 10, pp. 25–27; Berry et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory," pp. 105–109; and Berry and Lewinsohn, "Dose-Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards, Part I. Morbidity," pp. 187–191. See also, RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 66; and RCA Transcript, Evidence of Dr. Hans Weill, 16 June 1981, Volume no. 9, pp. 59–60.

[372] Berry and Lewinsohn, "Dose-Response Relationships for Asbestos-Related Disease: Implications for Hygiene Standards, Part I. Morbidity"; and Berry et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory."

I. What is the Health Risk from the Ingestion of Asbestos Fibres?

The principal method by which asbestos fibres enter the body is through inhalation. It is the inhalation of asbestos fibres which has been responsible for the disease incidence found in asbestos workers. But asbestos fibres may also enter the body through ingestion, either in food, beverages, drinking water, or drugs. Anyone in the general population from time to time may well ingest asbestos fibres from one of these sources. For that matter, asbestos workers may be prone to ingest asbestos fibres where food is eaten on the job and has been contaminated by the workplace environment.

It is therefore critical to consider whether there is a health risk from the ingestion of asbestos fibres. Of course, part of the answer to this question may turn on the sizes of asbestos fibres in food and drink and the amount of asbestos actually ingested. This we discuss in Chapter 11 where we provide detailed information on the sizes of fibres in food and drink. Here our inquiry is limited to considering whether there is any evidence that the ingestion of asbestos fibres is capable of producing a human health risk. If there is such a risk, it is likely to be related to malignancies of the gastrointestinal tract. Several cohorts of asbestos workers have indeed demonstrated an excess risk of gastrointestinal cancer from asbestos exposure. And while that excess risk has not been consistent from cohort to cohort — the Rochdale and Charleston textile workers, the New Orleans asbestos-cement workers, and the Nottingham gas mask workers being among the working populations where there was no excess gastrointestinal cancer — there is still a considerable body of evidence which points to an association between asbestos and cancer of the esophagus, stomach, colon, and rectum, albeit a much weaker association than, for example, between asbestos and lung cancer.[374]

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[373] See Peto, "An Alternative Approach for the Risk Assessment of Asbestos in Schools"; and see note 319, *supra*. Dr. Acheson testified that there is no evidence in the United Kingdom of mesothelioma having occurred as a result of exposure to asbestos in schools. See RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, pp. 28–29. Dr. Anderson testified that he was not aware of any mesotheliomas attributable to school asbestos exposure. See RCA Transcript, Evidence of Dr. Henry A. Anderson, 7 July 1981, Volume no. 16, p. 60. See also, RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 117.

Equally significant, that association arises from asbestos exposure through inhalation. There is, to our knowledge, no evidence that the direct oral ingestion of asbestos by itself is capable of producing an excess risk of gastrointestinal cancer. Admittedly, epidemiological methods are not overly sensitive to this issue — first, because it is difficult to find an appropriate control group for comparison purposes and second, because gastrointestinal tumours are much less specifically related to asbestos exposure than, for example, mesothelioma.[375] Yet the fact remains that at the present time the suggestion that eating or drinking asbestos fibres poses a health risk is not supported by any substantial evidence. Indeed, there are three pieces of evidence, apart from actual ingestion levels, that suggest a contrary conclusion. One is that, as Dr. Eric J. Chatfield has indicated, the sizes of fibres actually ingested in food or beverages or drinking water appear to be very small, with a mean length of 2 microns or less.[376] By comparison, as we have seen, the weight of the evidence indicates that it is the longer fibres, 5 or perhaps 8 or more microns in length, which are most hazardous from a health point of view.

A second piece of evidence is the general conclusion that emerges from the various studies that have considered asbestos concentrations in municipal water systems. While recognizing the limitations of epidemiology in addressing this issue, the fact remains that generally there do not appear to be any adverse health effects even where asbestos levels in water systems are thought to be elevated. A recent study by Toft et al. investigated mortality rates and asbestos levels in the drinking water systems of 71 Canadian municipalities and concluded there was no significant relationship between these levels and gastrointestinal cancer.[377] A similar conclusion was reached by Dr. Chatfield after analyzing asbestos levels in the drinking water supply systems of 18 municipalities in Ontario.[378] More recently, Dr. Jack Siemiatycki assessed the mortality experience of the general population in the mining area of Quebec.[379] He noted that Thetford Mines has one of the highest levels of asbestos fibre concentrations among North American drinking water systems and that levels at Asbestos, which has had a filtration system since the 1950s, are also relatively high and would have been higher prior to the 1950s. Table 5.35 shows mortality from selected causes for female residents of Asbestos and Thetford Mines for the period 1966–1977. Despite the fact that the drinking water in Asbestos and Thetford Mines may be as much as 100 times more polluted with chrysotile asbestos than the drinking water in other North American and European cities, the author could find no statistically significant excess mortality from this environmental exposure.[380] Indeed, of the several studies of the health risks from drinking water, only the report by Conforti et al. of the water supply system in the San Francisco Bay area even suggests a relationship between ingested asbestos and gastrointestinal cancer and the relationship is weak.[381] Only a fraction of the many analyses performed by Conforti et al. suggested a correlation of asbestos to cancer, and the authors noted that confounding factors such as smoking, occupation, and alcohol consumption may be important but were not allowed for in the study.

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[374] See Chapter 2, Section D.4 and Sections B and C.5 of this chapter.

[375] RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 95.

[376] RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, p. 97.

[377] Peter Toft et al., "Asbestos and Drinking Water in Canada," *The Science of the Total Environment* 18 (1981): 71-89.

[378] See RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, pp. 89-92; and Eric J. Chatfield and M. Jane Dillon, *A National Survey for Asbestos Fibres in Canadian Drinking Water Supplies*, 79-EHD-34 (Ottawa: Health and Welfare Canada, Environmental Health Directorate, 1979), pp. 43-44.

A final piece of evidence is the animal data, the bulk of which tends to demonstrate not merely the absence of malignant tumours, but an absence of asbestos fibres altogether in the gastrointestinal tract of rats following substantial feeding of asbestos.[382] For example, Dr. Davis has carried out a number of animal ingestion experiments on rats. He has found no evidence of asbestos retention within the gut, no sign of cell penetration or damage to the intestinal mucosa, and no evidence at all of gastrointestinal tumours. By contrast, Dr. Davis has pointed out that there is plenty of evidence of asbestos fibres in the gastrointestinal tract of rats following inhalation experiments.[383]

This animal evidence must be viewed with some caution. Although the weight of the animal data appears to be consistent with the findings made by Dr. Davis, a few experiments have indicated that small amounts of

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[379] Jack Siemiatycki, "Health Effects on the General Population (Mortality in the General Population in Asbestos Mining Areas)," in *Proceedings of the World Symposium on Asbestos*, Montreal, Quebec: 25–27 May 1982 (Montreal, P.Q.: Canadian Asbestos Information Centre [1983]), pp. 337–348.

[380] Ibid., p. 342.

[381] Paul M. Conforti et al., "Asbestos in Drinking Water and Cancer in the San Francisco Bay Area: 1969–1974 Incidence," *Journal of Chronic Diseases* 34 (1981): 211–224.

[382] RCA Transcript, Evidence of Dr. E. Donald Acheson, 20 July 1981, Volume no. 19, p. 115.

[383] RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 97–100; R.E. Bolton and J.M.G. Davis, "The Short-Term Effects of Chronic Asbestos Ingestion in Rats," *Annals of Occupational Hygiene* 19:2 (November 1976): 121–128; and J.M.G. Davis, R.E. Bolton, and J. Garrett, "Penetration of Cells by Asbestos Fibers," *Environmental Health Perspectives* 9 (1974): 255–260.

Table 5.35

Mortality, from Selected Causes, in Asbestos and Thetford Mines Compared to Quebec Province, Females, 1966–1977

Cause	O	E	O-E	L.C.L.*	O/E	U.C.L.*
All causes	1,130	1,274.6	-144.6	0.84	0.89	0.94
All cancers	289	318.1	-29.1	0.81	0.91	1.02
Digestive cancer	117	110.7	6.3	0.88	1.06	1.28
Respiratory cancer	23	21.5	1.5	0.68	1.07	1.61
Other respiratory disease	30	51.8	-21.8	0.39	0.58	0.83

Note: *95% confidence limits. ["L.C.L." means lower confidence limit; and "U.C.L." means upper confidence limit.]

SOURCE: Jack Siemiatycki, "Health Effects on the General Population (Mortality in the General Population in Asbestos Mining Areas)," in *Proceedings of the World Symposium on Asbestos*, Montreal, Quebec: 25-27 May 1982 (Montreal, P.Q.: Canadian Asbestos Information Centre [1983]), Table 6, p. 348.

asbestos following feeding may be found in intestinal tissues and have suggested an association between ingestion and gastrointestinal tumours.[384] Further, at the present it is not entirely clear as to whether the animal data are even applicable to humans for we are not certain as to how fibres reach the gastrointestinal tract or as to the mechanism by which asbestos apparently induces alimentary tract tumours in humans. We conclude on the basis of the evidence that the inhalation rather than the ingestion of fibres is responsible. One possibility is that asbestos fibres may reach the gastrointestinal tract by being coughed up and swallowed following inhalation.[385] Penetration of the gastrointestinal tract after swallowing would be far less likely since the fibres would tend to be "flushed" through the system. Another possibility suggested by Dr. Davis and more consistent with the animal data is that asbestos, after inhalation (but not ingestion), has the ability to move around the body by the lymphatic channels to various sites, including that of the alimentary tract.[386] However, Dr. Alexander C. Ritchie, Professor of Pathology at the University of Toronto, expressed serious reservations about this possibility in his testimony before the Commission. Dr. Ritchie had no doubt that asbestos fibres can find their way to the gastrointestinal tract following inhalation (and, if anything, he thought the bloodstream a more likely route), but he candidly observed that we really do not know how they get there.[387]

Accepting the uncertain state of medical knowledge on this matter, we cannot exclude completely the possibility of adverse health effects following the direct ingestion of asbestos. However, we deem it most unlikely. First, we believe it unlikely that asbestos fibres penetrate the gastrointestinal tract following ingestion. Second, even if they do, the fibres one ingests from non-occupational sources are very small, much shorter than the length we consider to be hazardous. Accordingly, it is reasonable to conclude that at present the evidence fails to indicate any increased risk of alimentary tract tumours following the direct ingestion of asbestos fibres.

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[384] A number of the animal studies have been reviewed in World Health Organization, *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*, vol. 14: *Asbestos*, pp. 58-60. See also, Davis, "The Use of Animal Inhalation Experiments in the Study of Asbestos Bioeffects," pp. 569-570.

[385] Kotin, "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," p. 138.

[386] RCA Transcript, Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, pp. 100-101.

[387] RCA Transcript, Evidence of Dr. Alexander C. Ritchie, 14 July 1982, Volume no. 49, pp. 14-19, 31-33. Dr. Ritchie testified that if one considers the hypothesis that fibres can travel to various sites of the body by the lymphatic channels, there are a number of problems. First, the lymph flow from the stomach to the lung is from the stomach upwards, so that one would have to imagine these inert asbestos fibres somehow swimming against the flow. Even if one allows that the fibres could proceed against the normal direction of the flow, if one takes the ordinary view that the most hazardous fibres are the long, thin ones, then these fibres would have to travel in vessels that are not much bigger than the fibres themselves, and certainly much narrower than 10 microns in length.

Volume

2

Ontario

Report of The Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario

Volume Two

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Part III

Asbestos in Fixed Workplaces

Chapter 6 Fixed Place Industry: Asbestos Use and Control

A. Introduction

Having explored the health effects of asbestos fibre exposure, we now turn to an examination of the uses of asbestos in Ontario, the exposure of workers to asbestos fibres, and the measures that must be taken adequately to protect the health and safety of Ontario workers. Workplace exposure can be divided into two broad categories, currently recognized by the Ontario Ministry of Labour: fixed place exposure and non-fixed place exposure.[1] Fixed workplaces include manufacturing operations, mining, shipbuilding and repair, railroad equipment maintenance, and automotive brake maintenance. Non-fixed workplaces include building construction, renovation or demolition projects. The exposure of workers in non-fixed workplaces, and in buildings, will be discussed in Part IV of this Report. The present Part deals with fixed workplaces.

We begin this chapter by presenting information on the level of activity in asbestos manufacturing in Ontario and in Canada, along with asbestos product trade statistics which indicate the economic activity that occasioned past and current worker exposures. We also summarize the exposure of Ontario workers to airborne asbestos since the mid-1950s. We then review the technology for controlling worker exposure to asbestos and present estimates of the cost implications for some of the major manufacturing activities that currently exist in Ontario. Because replacing asbestos with other fibres may be an important method of controlling worker exposure to asbestos, the cost and availability of asbestos substitutes are discussed. The health effects of the substitutes are then reviewed to determine whether we can have confidence that substitution will reduce, rather than increase, the risk to which Ontario workers are exposed. The final sections of this chapter deal with fixed workplace asbestos exposures other than in manufacturing. While mining of asbestos has never been a major activity in Ontario, we present a brief profile of asbestos and related mining in the province with estimates of the worker exposures that have resulted. We also review exposures in such domains as automotive brake repair, shipbuilding, and railroad equipment construction and maintenance.

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[1] *Occupational Health and Safety Act*, R.S.O. 1980, c. 321, s.1, par. 23.

In Chapter 7 we go on to examine the problems of measuring asbestos fibre exposures in fixed workplaces, and ways to improve current measurement methods. The health risks to Ontario workers from fixed workplace exposures are then estimated, relying on the health effects information developed in Chapter 5. The current regulations for worker exposure in fixed workplaces in Ontario and elsewhere are reviewed and the control limits that should govern worker exposure to asbestos in Ontario are considered and recommended. Work practices necessary to protect workers in fixed workplaces where monitoring is not feasible are also discussed.

Chapter 8 moves beyond numerical control limits to the implementation of controls in fixed workplaces. The operations of the *Occupational Health and Safety Act* and its subordinate Regulation Respecting Asbestos are explored, weaknesses identified, and recommendations for improvements made. This chapter expresses our conviction that control limits only create an illusion of healthy workplaces in the absence of implementation measures bent on actually achieving those control limits, and moving beyond them where practicable.

B. Profile of the Canadian Asbestos Industry

B.1 Asbestos Manufacturing in Ontario

Determining which firms in Ontario belong to the asbestos manufacturing industry is difficult because the industry must be defined on the basis of an input that it purchases rather than on the basis of the type of output it produces. The firms classified as belonging to the asbestos industry will use asbestos in a wide variety of products in widely varying amounts, and may, save for the use of asbestos, be completely unrelated.

A written submission made by the Ontario Ministry of Labour to this Commission identified 15 firms as the asbestos manufacturing industry in Ontario.[2] However, two of those firms had discontinued the use of asbestos and one had abandoned manufacturing and begun to engage in asbestos insulation removal. Thus, this list includes at best 12 major manufacturers of asbestos products. To supplement this list, the Ministry of Labour surveyed its Industrial Health and Safety Branch files of 191 companies whose workers were under surveillance by the Occupational Health Branch for use of asbestos. Of these 191 companies, 90 reported recent asbestos exposure.[3] From the list of 90 asbestos users, we eliminated firms that consumed asbestos in only marginal quantities, or were involved in the installation or modification of a product containing asbestos and not its actual manufacture. Combining the remaining firms with the firms identified in the Ministry of Labour submission yielded the list of 23 firms in Table 6.1. The diversity of the industry is readily apparent from examining the products manufactured and the number of exposed employees. These firms do not constitute a single homogenous industry. Products manufactured range from asbestos-cement pipe and construction products to brake linings. Save for the use of asbestos as an input, the firms in the industry are completely unrelated. There is no industry association to which most of the members of this group belong. Neither is there any general contact among most members of the group.

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[2] Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, p. 7.

Table 6.1 indicates whether the manufacturing firm is owned by another company and the country of origin for the parent company. Of the 23 firms listed in the table, slightly more than half are controlled by companies outside of Canada, all but two of which are located in the United States. In cases of foreign control, the parent firm almost always controls 100% of the subsidiary company. Typically, the foreign firm has not one but several Canadian subsidiaries, sometimes producing in different provinces, sometimes dividing responsibility for production, sales, distribution, and other functions. The other firms in the Canadian family usually do not produce asbestos-containing goods. However, it is not uncommon for firms in the family outside of Canada to be involved in some type of asbestos-related activity.

In addition to the number of Canadian firms owned by the parent, Table 6.1 provides information on the assets of the Canadian asbestos manufacturing firms where this information is available. The asset information is not representative because it can only be obtained for the largest firms. There are no readily available public records of the assets of the smaller domestic firms. Aggregate data, however, show that for 1981, the asbestos manufacturing firms listed in Table 6.1 had assets totalling

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[3] Ibid. See also Appendix 6 to the Ministry of Labour's written submission to the Royal Commission on Asbestos, "Companies Using Asbestos and Under Medical Surveillance by the Occupational Health Branch, Updated September 22, 1980," Appendices, pp. 31-37; and revised list issued by letter from Mr. Arthur L. Gladstone, Senior Policy Advisor, Occupational Health and Safety Division, Ontario Ministry of Labour to the Royal Commission on Asbestos, 6 June 1983.

Table 6.1
Ontario Asbestos Product Manufacturers
(1981 Data Except as Noted)

Company	Product	Assets ($ Millions)	Workers Under Surveillance, 1980*	Parent Company			
				Name	Country	% Owned	Number of Firms in Family**
Abex Industries Ltd.	Friction materials	50.4	134	I.C. Industries Inc.	U.S.A.	100.0	15
Able Gasket and Materials Ltd.	Gaskets		15		Canada		
Acro Gasket Industries Ltd.	Gaskets		23				
BBA Automotive Ind. (Cdn.) Ltd.	Brake parts	7.9	83	BBA Group Ltd.	U.K.	100.0	2
Bakelite Thermosets Ltd.	Thermosets		20	Lockston Ind. Ltd.	Canada	64.7	2
Bendix Auto of Canada Ltd.***	Friction products	65.4	460	The Bendix Corporation	U.S.A.	100.0	13
Canadian Cylinder Company Ltd.	Absorbant in acetylene cylinders		19	Pacific Lumber Corp.	U.S.A.	100.0	6
Cataract Canvas Ltd.			NA†		Canada		
Certified Auto. Prod. Ltd.	Friction products		316	Lear Siegler Inc.	U.S.A.	100.0	6
A.W. Chesterton Co.	Valve packing asbestos yarn		2	A.W. Chesterton Co. Inc.	U.S.A.	100.0	1
Durable Canada Ltd.	Gaskets		11		U.S.A.	100.0	
The Flintkote Co. of Canada Ltd.	Joint compound		13	Genstar Ltd.	Belgium	100.0	113
Garlock of Canada	Braids and sheets	19.1	50	Colt Industries	U.S.A.	100.0	8

Table 6.1 (continued)
Ontario Asbestos Product Manufacturers
(1981 Data Except as Noted)

Company	Product	Assets ($ Millions)	Workers Under Surveillance, 1980*	Parent Company			
				Name	Country	% Owned	Number of Firms in Family**
Hill Machine and Asbestos			11				
Inmont Presstite Ltd.	Sealant	1.7	7	United Technologies Corp.	U.S.A.	99.2	21
Insul Coustics			NA				
Johns-Manville Canada Inc.***	A/C pipe	380 (1979)	500	Johns-Manville Corp.	U.S.A.	100.0	8
PRC Chemical Corp. of Canada Ltd.	Sealants	4.9	25	Products Research and Chemical Corp.	U.S.A.	100.0	3
Ranger Safety Products	Asbestos cloth, gloves		19				1
Raybestos-Manhattan (Can.) Ltd.	Friction materials	10.3	126	Raybestos-Manhattan Inc.	U.S.A.	99.2	7
Reichhold Ltd.	Mouldings		16	Reichhold Ltd.	Canada		
Relmech Mfg. Ltd.	Electric insulators		38				
Scott Laboratories	Filter pads		10				

Notes: *Workers under surveillance by the Ministry of Labour's Occupational Health Branch.
**In Canada excluding listed company.
***Not active in asbestos product manufacture in 1981.
†NA means data not available.

SOURCE: Statistics Canada, Ontario Ministry of Labour, *Moody's Industrial Manual.*

$731 million, equity of $449 million, and $69.8 million in profit on sales of $870 million. These aggregate assets included the remaining assets of Bendix and Johns-Manville, both of which ceased asbestos operations in Ontario in 1980.

The third column of Table 6.1 which is headed "Workers Under Surveillance, 1980' can be taken, with the exception noted below, as a rough proxy for the number of workers in asbestos exposure employment in each firm during that year. The data were supplied to us by the Ministry of Labour and show the number of individuals under medical surveillance by the Ministry's Occupational Health Branch. The data are a proxy for asbestos exposure employment because ex-employees are not notified of the visits made by the Ministry's mobile x-ray units, save at Johns-Manville whose figure of 500 therefore overestimates the size of its workforce. (See Chapter 14, Section B.) With this exception, the data illustrate by firm the recent levels of asbestos manufacturing activity and prevalence of exposed employees. These evidently vary greatly, from 2 exposed employees at A.W. Chesterton to 480 at Bendix. Bendix, like Johns-Manville, ceased asbestos operations in 1980. It is apparent from Table 6.1 that, of the firms that remain active, friction product manufacturers account for about two-thirds of some 1,000 employees exposed to asbestos in Ontario manufacturing.

To gain a perspective on the changes in the structure of the industry over time, data were obtained from the Statistics Canada Census of Manufactures. For the years 1952 to 1969, the statistics were taken from an annual publication. However, for subsequent years statistics were developed specifically for this Commission. The criterion we adopted for defining membership in the industry was that 50% of the value of the total output of each firm be composed of products which were primarily composed of asbestos. This yielded a varying list of up to 10 firms, most of which are included in the list of Table 6.1. However, since employers have the discretion to classify their products as primarily composed of asbestos, or not, the accuracy of this industry definition is affected by the great latitude that firms enjoy in classifying their products. For instance, friction product manufacturers are included in this industry, although their final products may contain as little as 10 to 25% asbestos by weight. Table 6.2 shows that the industry experienced significant changes in the last decade. In 1980, the industry was composed of 10 firms and had shipments and revenues of asbestos products and non-asbestos products valued at $109 million. This represents a 62% nominal increase in the 6-year period from 1974 to 1980, and a 9.6% decrease in real terms after adjusting for inflation.[4] The total nominal value of asbestos products (not shown in Table 6.2) rose by $32.7 million or 96% in the same period. Part of this increase is undoubtedly attributable to the change in the number of establishments in 1979. However, the increase also reflects the changing composition of the products manufactured by the industry; between the years 1974 and 1980, the value of friction products produced in Ontario increased by 300%, while in the same period the value of goods produced in the asbestos-cement products and asbestos tile categories declined by 19%. The reduction in manufacturing activity in the asbestos-cement product category caused consumption of crude asbestos in the industry to fall dramatically in a 6-year period. In 1974, the industry consumed 16.1 thousand tons of asbestos fibre with the value of $4.1 million. However, by 1980, the quantity of asbestos declined by 50% to 8,000 metric tons with a value of $4.0 million, reflecting a decline in the asbestos-cement product category. Asbestos represents a smaller percentage of total product value in friction products than in asbestos-cement, so the growth in friction products did not offset the contraction of activity in the asbestos-cement product sector. Behind the contraction of the asbestos-cement product sector evidenced in Table 6.2 lies the closure of the Johns-Manville Canada pipe operation in 1980 and the reduction in demand for asbestos-cement products by the depressed domestic and international construction industries.

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[4] This index is based on price trends experienced by asbestos-cement products and friction products which amounted to a 79% increase between 1974 and 1980.

It is possible that in addition to the general decline in activity in the construction sector, the reduced demand for asbestos-cement products is also a function of the increasing concern with the health effects of asbestos and the availability of cost- and performance-competitive substitute materials. The increase in friction product activity in Ontario may perhaps be attributable to the growing reliance of U.S. brake manufacturers on their Canadian operations to escape from the burgeoning number of asbestos-related tort claims being brought by workers suffering asbestos disease in the United States.

Professor Gordon M. Bragg conducted a survey for this Commission of the 23 firms listed in Table 6.1, gathering data which are in general agreement with the principal statistics from Statistics Canada reported in Table 6.3. Professor Bragg reported asbestos use by the firms in Ontario, not including Johns-Manville, for the year 1980 as shown in Table 6.4.

The dominance of friction products manufacture over the rest of the industry is clear as friction products account for over 50% of asbestos use in 1980. However, five years earlier the asbestos use by friction manufacturers was dwarfed in comparison to Johns-Manville's consumption: fully 75% of the total asbestos input to the Ontario industry was consumed at its plant.

Included in Professor Bragg's survey were responses to questions concerning the competitive conditions under which the asbestos products manufacturers in Ontario operate. Of the 10 firms which responded to the question concerning competition in Ontario, elsewhere in Canada, in the

Table 6.2

Principal Statistics, Asbestos Manufacturing: Ontario*

	1962	1956	1960	1964	1969	1974	1977	1979	1980
Number of establishments	8	8	9	8	7	9	9	10	10
Value of shipments ($ Millions)	NA	NA	12.6	22.1	28.5	67.4	79.4	113.8	109.3
Manufacturing value added ($ Millions)	NA	9.8	7.2	13.0	18.0	32.2	41.3	63.4	57.5
Total value added ($ Millions)	NA	NA	NA	13.3	19.1	35.5	41.5	66.9	58.2
Quantity of asbestos fibre (000 metric tons)	NA	NA	NA	NA	NA	16.1	12.5	13.4	8.0
Value of asbestos fibre ($ Millions)	NA	NA	NA	NA	NA	4.1	6.6	6.9	4.0
Employees administration Employees production	423	785	784	237 938	366 923	383 1,389	319 1,199	375 1,345	385 1,195
Total salaries and wages ($ Millions)	1.4	3.3	3.6	NA	9.8	18.2	21.4	26.7	27.3

Note: *Industry includes only firms for which over 50% of total product value is products containing asbestos.

SOURCE: Statistics Canada.

Table 6.3

Principal Statistics, Asbestos Manufacturing: Canada*

	1952	1956	1960	1964	1969	1974	1977	1979	1980
Number of establishments	17	16	17	16	15	12	12	16	16
Value of shipments ($ Millions)	19.6	34.2	29.4	43.0	50.3	112.1	135.3	193.3	184.0
Manufacturing value added ($ Millions)	NA	18.4	15.8	24.9	31.1	52.2	66.0	96.7	85.7
Number of production employees	1,826	2,249	2,086	2,570	2,596	2,091	1,961	2,123	1,835
Quantity of asbestos fibre (000 metric tons)	44.6	72.8	54.3	60.7	67.6	50.2	47.4	55.3	36.7
Value of asbestos fibre ($ Millions)	4.0	5.4	4.9	5.3	3.8	11.1	17.0	21.0	15.1
Average weekly earnings ($)	NA	71.13	79.19	94.97	128.93	176.46	255.74	305.14	321.66

Note: *Industry includes only firms for which over 50% of total product value is products containing asbestos.

SOURCE: Statistics Canada.

Table 6.4

Asbestos Use in Ontario Manufacturing

Products	**Asbestos Used (Metric Tons/Year)**
Friction products	4,500
Floor tiles	1,000
Gaskets and packings	1,250
Caulking, sealants, coatings, and paints	600
Plastics	400
Cylinders	180
Insulators	100

SOURCE: Gordon M. Bragg, *The Technical Feasibility and Cost of Controlling Workplace Exposure to Asbestos Fibres,* Royal Commission on Asbestos Study Series, no. 7 (Toronto: Royal Commission on Asbestos, 1982), p. 7.

United States, or in the world, seven reported competition from the U.S. and three from other foreign countries. Vagt has reported that most manufactured asbestos products from other countries face a most favoured nation tariff of 11.9% (United States, West European countries), a general tariff of 25% (East European countries), or a general preferential tariff of 7.5% (underdeveloped countries).[5] Products made from crude asbestos of British Commonwealth origin are free under the British Preferential Tariff. Thus, it seems plausible that Canadian manufacturers of friction products must compete openly with each other and with any other U.S. firm, given the trade arrangements of the auto pact. The floor tile, gaskets and packing, caulking, sealants, coatings, and paints, and the plastics sector seem to face some domestic competition, and U.S. competition which is limited only by the tariff.

The Bragg survey enquired about the anticipated demand for asbestos fibre in the next 10 years. Apart from the friction product manufacturers, most companies responding expected that their use of asbestos in product manufacture would be discontinued by 1987. Furthermore, most of these manufacturers felt that they would produce a non-asbestos product with similar cost and performance characteristics which would substitute for the asbestos product. However, none of the friction product manufacturers, which constitute the largest segment of the Ontario asbestos manufacturing industry, foresaw their use of asbestos being curtailed by 1987.

The declining importance of asbestos as an input for non-friction product manufacturers was also confirmed by the Ministry of Labour investigation of asbestos users. Of the firms which were under surveillance by the Occupational Health Branch for asbestos use during the 1970s, 21% had by the end of the decade discontinued their use of asbestos as an input.

B.2 Asbestos in Canada

How does the structure and activity of the Ontario manufacturing industry compare to that of the Canadian asbestos manufacturing industry as a whole? Employing the same method that was used to generate Table 6.2, Statistics Canada provided primary statistics for the Canadian asbestos manufacturing industry as a whole. In 1980, the total asbestos input to Canadian manufacturing was 36.7 thousand metric tons, almost precisely the Vagt estimates. (See Table 6.3.) Six firms outside Ontario were considered asbestos product manufacturers, generating $74.7 million in total shipments and revenues, not shown in Table 6.3. This amount constitutes 41% of the total Canadian shipments and revenues of $184 million. However, the asbestos used by the six non-Ontario firms constituted 73%

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[5] G. Oliver Vagt, *Asbestos* (Hull, Quebec: Supply and Services, 1980), p. 11.

of the total asbestos input in the entire industry. Again, this disparity may be traced to the concentration of the Ontario firms in friction products manufacture, while the non-Ontario firms were involved primarily in activity in the asbestos-cement pipe and building products sector.

While Ontario's involvement with asbestos is overwhelmingly as a manufacturer and consumer of asbestos products, other provinces are active not only in asbestos products manufacture, but in production of crude asbestos as well. World production of asbestos was about 5 million metric tons in 1980, of which about 90% was chrysotile.[6] In 1980, Canada shipped 1.335 million metric tons of chrysotile valued at $642 million. Approximately 88% of this production was from Quebec, 7% from British Columbia, and 5% from Newfoundland.[7] Vagt showed Canadian asbestos consumption at 0.7% of world consumption, or approximately 36,000 metric tons.[8]

Canadian imports and exports of asbestos and asbestos products are shown in Table 6.5. During the last few years, Canada has imported considerable quantities of manufactured asbestos products including brake linings, packing materials, textiles, clutch facings, and asbestos-cement building materials. While a breakdown of asbestos products imported into Ontario is not available, we have identified the amount of South African asbestos, presumably crocidolite, that has been imported into Ontario. In 1979, this was 209 tons; in 1980, 230 tons; and in 1981, none. It is believed that the cessation of crocidolite importing into Ontario after 1980 is a reflection of the closing of the Transite pipe section of the Johns-Manville plant in Scarborough, which was a major user of crocidolite.

Table 6.5 also shows exports of asbestos fibre and products from Canada. The export of brake linings is about three-quarters the imports of those linings, reflecting the two-way trade with the United States occurring under the auto pact. The most important single manufactured product that is exported is asbestos-cement building materials valued at $16.9 million in 1981.

The structure of Canadian asbestos manufacturing can be compared with that of the U.S. industry. Principal statistics on the asbestos manufacturing industry in the United States indicate total shipments and revenue of $1,029.9 million U.S. ($1,203.9 million Canadian, 1980). Of that amount 34.3% of the shipments and revenues accrued to friction products manufacture, 32.9% to asbestos floor tiles, and 31.3% to asbestos textiles, insula-

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[6] Ibid., p. 7.

[7] Ibid., p. 1.

[8] Ibid., p. 7.

Table 6.5

Canadian Trade in Asbestos Fibre and Asbestos-Based Products
(\$ Millions – Current)

Imports		1972	1974	1977	1979	1980	1981
Number	Description						
47420	Asbestos textiles	1.1	3.0	3.9	2.8	2.4	1.9
47424	Asbestos packing	1.1	1.9	2.1	2.9	3.2	3.5
47437	Asbestos brake linings	4.9	5.3	4.8	7.8	6.6	8.3
47438	Asbestos clutch facings	0.6	1.4	1.3	1.7	1.6	1.5
47464	Asbestos pipe cover	0.2	0.1	0.1	0.03	0.06	0.06
47466	A/C board/sheets	0.8	0.7	0.5	0.6	0.8	0.5
47469	A/C building materials	3.8	8.2	5.9	4.5	3.5	2.2
47499	Asbestos basic products	1.9	2.6	3.8	6.1	5.2	3.5
27140	Milled asbestos fibres			2.0	1.0	0.9	0.7
Exports							
Number	Description						
23110	Asbestos crude	0.132	0.08	0.003	0.012		0.026
22120	Asbestos milled fibres, Group 3	13.7	21.9	27.6	32.0		18.8
27130	Asbestos milled fibres, Groups 4 and 5	156.6	227.8	393.0	465.0		424.4
27140	Asbestos shorts, Groups 6-9	64.7	89.5	132.9	155.6		131.6
47420	Asbestos textiles		3.2	1.2	6.4		4.0
47440	Asbestos brake linings	0.8	1.3	3.3	3.4		6.3
47469	A/C building materials	2.2	5.2	15.0	16.8		16.9
47499	Asbestos basic products	2.4	3.5	5.7	10.1		9.4

SOURCE: Statistics Canada, Asbestos Mines: Mineral Statistics.

tion, and asbestos-cement products.[9] In Ontario, by contrast, Professor Bragg has reported that 78% of the shipments and revenues of the asbestos industry accrued from friction products manufacture.

B.3 Ontario Worker Exposure to Asbestos

What worker exposure to asbestos fibres has resulted from asbestos product manufacturing in Ontario? Most available data were gathered after about 1970, when the membrane filter method was adopted for widespread use in monitoring occupational exposure to asbestos fibres. During the 1950s and 1960s, the Bausch and Lomb dust counter was used along with the Greenburg-Smith impinger, both of which measure dust levels in millions of particles per cubic foot. Very limited data indicate that manufacturing dust counts measured by the Bausch and Lomb dust counter averaged between 10 and 25 million particles per cubic foot (mppcf), with a range of values as high as 50, 75, or even 100 million particles per cubic foot in some samples. Particle counts taken with the midget impinger during the 1950s and 1960s averaged generally between 4 and 10 million particles per cubic foot, with the maximum concentration in individual samples ranging to 20, 30, or as much as 60. (See Table 6.6.) One source in 1958 produced an average of 32 million particles, with the highest single sample reading 134 million particles using the midget impinger. Table 6.6 summarizes the midget impinger data for firms producing friction products, asbestos-cement (A/C) pipe, and miscellaneous products.

By 1970, the particle counts using the midget impinger were declining, and this measurement was replaced by the membrane filter method which measures the concentration of fibres. Table 6.7 shows average annual fibre counts using the membrane filter method in five friction products plants, three gasket plants, and one asbestos-cement pipe plant in Ontario. Each average is an average of all area and personal samples in all plants monitored during that year. The percentage of all samples in which the count exceeded 2 fibres per cubic centimetre (f/cc) is also shown. The great variability in data such as these is discussed in a study prepared for this Commission by Dr. Eric J. Chatfield and in Chapter 7, Section B of this Report.[10] These data show generally declining fibre counts during the 1970s, with many plants achieving levels well below 1 fibre much of the time in the late 1970s. It appears that friction products have higher dust levels than either gasket and packing or asbestos-cement operations.

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[9] Telephone communication between Mr. John Ambler, U.S. Bureau of Census, Washington, D.C. and Royal Commission on Asbestos Staff. Data are for Standard Industrial Classification 3292.

[10] Eric J. Chatfield, *Measurement of Asbestos Fibre Concentrations in Workplace Atmospheres*, Royal Commission on Asbestos Study Series, no. 9 (Toronto: Royal Commission on Asbestos, 1982), sec. 2.4.

Table 6.8

Summary of Asbestos Dust Levels in Ontario Manufacturing Measured by Midget Impinger*
(Millions of Particles Per Cubic Foot)

	Friction Products		Miscellaneous		A/C Pipe	
Year	Average	Range	Average	Range	Average	Range
1956					8.5	1.1-60
1958			32	2.1-134		
1961					7.8	0.4-28
1965	1.2	0.3-3.5				
1967	5.7	3.1-8.4				
1968					3.4	1.5-5.8
1969					7.9	6.5-9.2
1971			7.4	7.4	5.8	2.2-20
1972	11.7	6.0-30	2.0	1.0-3.7	2.2	0.5-5.8
1973					2.8	0.5-8.7
1974	4.2	1.4-7.6			4.4	1.5-32

Note: *Different plants may be included in the measurements of different years

SOURCE: Ontario Ministry of Labour, Occupational Health and Safety Division.

Table 6.7

Summary of Asbestos Dust Levels in Ontario Manufacturing Measured by Membrane Filter (Fibres per Cubic Centimetre)

Product:	Friction Products*		Gaskets and Packings*		A/C Pipe			
					Ministry Data*		Company Data (Personal Samples)**	
							Wet End	Finishing End
Year	Average	% Samples Greater than 2 f/cc***	Average	% Samples Greater than 2 f/cc***	Average	% Samples Greater than 2 f/cc***	% Samples Greater than 2 f/cc***	% Samples Greater than 2 f/cc***
1969	1.9	33	4.3	50	5.7	40	89	92
1970	4.1	75	NA†	NA	NA	NA	62	51
1971	3.6	63	1.2	0	1.0	0	46	31
1972	3.6	73	2.2	40	1.1	3	67	35
1973	1.6	30	NA	NA	1.7	25	8	8
1974	1.3	21	NA	NA	1.6	51	18	6
1975	1.3	16	1.1	24	0.6	0	16	4
1976	1.5	15	NA	NA	0.6	10	0	2
1977	2.9	36	1.2	0	0.5	0	0	0
1978	2.5	43	2.8	22	0.7	11	0	4
1979	1.6	23	0.4	0	0.3	5	0	0
1980	1.4	20	0.5	6	0.1	0	NA	NA
1981	0.7	10	0.1	0	Plant closed			

Notes: *Ontario Ministry of Labour, Occupational Health and Safety Division.
**From Johns-Manville company records.
***In 1972, the Ontario Ministry of Labour began to apply a guideline of 2 f/cc.
†NA means data not available — no readings taken.

SOURCE: See Notes * and ** above.

Unfortunately, it is not possible to convert the earlier particle counts to an equivalent fibre count using today's measurement technology in a general way, although some comparison studies have been conducted.[11] Thus, we cannot be sure how today's exposure levels compare with those of the 1950s and 1960s. While there is nearly universal agreement that the recent fibre measurements reflect dust particle levels far less than those prevailing in the 1950s, considerable disagreement remains as to what those levels actually were.

Dr. Murray M. Finkelstein reviewed the history of the Transite pipe section of the Johns-Manville plant and concluded that as compared to the years 1969–1970, exposures were 30% higher during 1955 through 1962, and were twice as high during 1948–1954.[12] By the late 1970s, exposures were reduced to generally less than 10% of the exposures of 1969–1970, depending on the work station. This yields estimated average exposure levels in three areas of the plant of 40, 16, and 8 f/cc in 1948–1954; 26, 10, and 5 f/cc during 1955–1962; and 20, 8, and 4 f/cc by 1969–1970. Finkelstein noted that all of these estimates are subject to considerable uncertainty, perhaps erring by a factor of 3 or 5 either way.

Rajhans, Bragg, and Morton surveyed Ontario Ministry of Labour records of membrane filter fibre counts at one asbestos mill, three friction product plants, and one asbestos-cement pipe plant.[13] The average readings for all plants declined from almost 4 f/cc in 1972 to about 1 f/cc in 1974, then rose close to 2 f/cc by 1976. All brake plants produced averages below 1 f/cc at some time, but two of the plants experienced considerable increases in fibre levels within a year after low readings were achieved. This survey concluded that a 2 f/cc standard was achievable in practice in 1977.

C. Technical Feasibility and Cost of Dust Control

C.1 Control Methods

Professor Bragg identified some basic methods of controlling the asbestos fibre exposure of workers.[14] The most important methods are:

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[11] See the discussion of early fibre estimates in Chapter 5, Section D, and of measurement techniques in Chapter 7, Section B.

[12] Murray M. Finkelstein, "Asbestosis in Long-Term Employees of an Ontario Asbestos-Cement Factory," *American Review of Respiratory Disease* 125 (1982): 497. See also, Chapter 3, Section A.3 of this Report.

[13] Gyan S. Rajhans, Gordon M. Bragg, and J. Stewart Morton, "A Review of Asbestos Exposure in Ontario," *American Industrial Hygiene Association Journal* 39 (September 1978): 767–771.

[14] Gordon M. Bragg, *The Technical Feasibility and Cost of Controlling Workplace Exposure to Asbestos Fibres*, Royal Commission on Asbestos Study Series, no. 7 (Toronto: Royal Commission on Asbestos, 1982), p. 34.

Substitution — Replacing the asbestos with a non-asbestos substitute in a product is a certain way of eliminating asbestos fibre exposure. A later section of this chapter will discuss the feasibility of substitution for asbestos in various products, and the possible health effects of substitutes.

Process Alteration — Some processes are inherently less dusty than others. Thus, for example, cutting asbestos-cement sheet with a knife creates less dust than sawing the same sheet.

Isolation and Enclosure — Placing a barrier between the worker and the source of asbestos dust, particularly by building an enclosure around the machine, process, or operation, can be highly effective in reducing the exposure of the worker. Local exhaust is usually used to prevent fibre release from the enclosure. Hazards still exist, however, when the enclosure is opened for feeding the machine or for maintenance.

Wet Methods — Soaking the asbestos product in a liquid, or spraying water on a cutting process, can dampen the fibres and thus prevent them from becoming airborne. Wet methods are highly effective at reducing dust, but may adversely affect the manufacturing process for some products.

Local Exhaust — Exhausting contaminated air from the region immediately around the process is the traditional method of dealing with asbestos dust problems. The exhausted air must be cleaned, typically in a bag-house, after which it may be re-used or blown outside. Moving this air, and warming the replacement air in winter, are costly in the Canadian environment.

Personal Protection Devices — For asbestos, the basic personal protection device is a respirator. While some models are quite effective, they are uncomfortable and inconvenient to wear for long periods of time.

Housekeeping — Regular careful cleaning of the workplace, including vacuuming rather than sweeping dust from floors, walls and ceilings, cleaning up spills when they occur, and maintenance of dust control equipment, can considerably reduce fibre levels, particularly when ventilation has already achieved reasonably low levels.

Monitoring Alarms — While some hazardous substances can be monitored continuously, so that any escape can trigger an alarm that will allow employees to avoid the substances, such monitoring is difficult and expensive for asbestos fibres, and there are serious problems with accuracy of the instrument. (See Chapter 7, Section B of this Report.)

Training — Where proper work practices and housekeeping are important for controlling dust levels, the training of employees in these techniques may significantly reduce employee exposures.

C.2 Feasibility of Control

The feasibility and cost of controlling fibre levels in the workplace can be evaluated by identifying operational processes where fibres may be released, and dealing with each of these separately. An asbestos product manufacturing plant will typically include a number of different processes, each of which may cause greatly different fibre releases. Since each process must be individually controlled, it is more accurate to analyze the processes separately than to compare one entire plant, such as a brake plant, with, for example, an asbestos-cement pipe plant.

Professor Bragg identified nine major operational processes that are typically used in Ontario asbestos manufacturing plants. These are listed in Table 6.8 along with the typical fibre levels found in Ontario in 1980 and 1981. Table 6.8 demonstrates that debagging is the dirtiest process, given current control technology, while cutting is relatively clean. For all of these processes, a change in the control technology could presumably change the fibre levels that are experienced by the operator.

Professor Bragg discussed the feasibility of controlling fibre levels in seven of the processes listed in Table 6.8. In doing so, he noted that there is considerable variability in control practices from one plant to another, depending in part on the level of engineering expertise within a particular company. He stated:

> The detailed application of engineering controls requires an intimate knowledge of ventilation practices, plant processes, and air pollution technology. We would estimate that there are fewer than twenty-five people within the province who could be considered truly expert in this field, most of whom are employed in specific plants. The others work in government or as consultants. In addition, the person must have an intimate knowledge of each plant to which he intends to apply controls. As may be suspected, the larger the plant, the higher the probability that this expertise exists within the company. The result of this process is that some companies have achieved higher control capability with less money and less interference with processes than others. In addition, we have found small manufacturing facilities with no engineering skills and little knowledge of control processes who are spending money but achieving very poor control. We see this latter problem with smaller industries as an important aspect in achieving province-wide controls.[15]

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[15] Ibid., p. 49.

Table 6.8

Typical Ontario Processes and Fibre Levels*

Operations Typical to Ontario Asbestos Industry	Typical Fibre Levels Found
	(f/cc)
Debagging	0.5 - 8.0
Pressing or forming	0.5 - 3.0
Grinding	0.5 - 5.0
Drilling	0.5 - 2.0
Cutting – Press	0.1 - 0.7
– Hand	0.2 - 2.0
Braiding	0.1 - 3.0
Spinning	0.1 - 3.0
Twisting	0.1 - 3.0
Sheeter operation	0.4 - 2.0

Note: *Based on 1980 and 1981 Ontario Ministry of Labour reports.

SOURCE: Gordon M. Bragg, *The Technical Feasibility and Cost of Controlling Workplace Exposure to Asbestos Fibres,* Royal Commission on Asbestos Study Series, no. 7 (Toronto: Royal Commission on Asbestos, 1982), Table 3.3, p. 47.

Professor Bragg also pointed out that a given fibre level cannot be achieved all the time, since even a clean process may occasionally produce high fibre levels.[16] High fibre levels are often released during the maintenance of machinery, failure of machinery, failure of air-conveying systems, worker sabotage of ventilation systems, improper usage of equipment, and high winds affecting the air cleaning system. There is some ambiguity about whether the fibre levels referred to in the Bragg study should be interpreted as average exposure levels, or as achieving compliance with a control limit, which would imply average exposures of half the control limit. In his discussion of control costs, the proper interpretation is as follows: references to fibre levels of 2, 1, and 0.5 f/cc are references to compliance with control limits of these amounts, so that average exposures are half the stated amounts; references to fibre levels of 0.1 f/cc refer only to the average exposure, and do not assure that compliance with a 0.1 f/cc control limit is possible. Professor Bragg's analysis can accordingly be seen as examining compliance with control limits of 2, 1, 0.5, and 0.2 f/cc and achieving average exposures of 1, 0.5, 0.25, and 0.1 f/cc.

Finally, Bragg concluded that higher levels of control are not always more expensive. For example, some enclosures use very little ventilating air, so the added expense of enclosure can, to some extent, be balanced against the decreased use of ventilating air.

Emptying asbestos out of bags, or *debagging*, is one of the most difficult processes to control. In this process the operator slits open 2- or 3-foot long plastic bags of asbestos and dumps or shakes the asbestos into a drum or hopper. A large working area is needed to accomodate the large bags. Since the working area is large, a large amount of ventilation air must be used. The empty bag may still contain considerable amounts of asbestos fibre, so placing the bag in a refuse container may also release a lot of dust. While several automated debagging machines are available, they cost about $50,000 each, and Bragg had serious doubts about their reliability and effectiveness.[17] He reported that none of these machines is in use in Ontario, although they would be necessary to reach 0.5 f/cc.

Professor Bragg reported that *grinding* and finishing produce large amounts of fibrous material presenting considerable control problems. Proper positioning of the air intake is imperative if 0.5 f/cc is to be achieved. Special machine designs dedicated to a specific grinding operation are necessary to achieve low fibre levels.

The control of fibre releases from *drilling* can be examined in the control of friction products manufacture. Drilling holes in the brake pads or linings is commonly done with a group of synchronized pneumatic drills. Good ventilation will ordinarily hold fibre levels to around 1 f/cc. Poor ventilation can allow the fibre levels to exceed 2 f/cc. If exhaust from the pneumatic system is allowed to blow on the ensuing dust, then fibre levels can rise well above 5 f/cc. Control to the 1 to 2 f/cc level commonly requires suction around the bottom of the drills to collect chips and dust and frequent vacuum cleaning of spilled material. Enclosing this process will require a device for feeding the brake linings or pads into the enclosure. Depending on the degree of automation this may require opening of the enclosure to feed-in new parts and to remove completed parts. Bragg estimated that a well-designed and enclosed automated process should reduce fibre levels down to 0.1 f/cc or lower.[18] For a semi-automated process where handloading is required, the fibre levels in the room would be below 0.1 f/cc during operation, and a sufficiently large exhaust would probably maintain this level during the opening of the enclosure. Enclosing a semi-automated process where handloading is used would result in a loss in output per worker in the order of 5 to 10%. The machine would remain as productive, but the amount of labour required would increase by this amount.

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[16] Ibid., pp. 49–50.

[17] Ibid., pp. 58–59.

Another process used in Ontario is the *cutting* of gasket material. Professor Bragg reported that hand-cutting of asbestos gaskets for custom installations results in a very small quantity of dust, usually around 0.1 to 0.7 f/cc.[19] Thus, no controls are required unless it is desired to reduce average exposures below about 0.5 f/cc. Cutting these gaskets on a table whose surface has a fine array of holes, and pulling air downwards through the table and away from the worker's face, would almost certainly provide extremely low dust levels. A more difficult control problem is presented by the sawing of large sheets of asbestos-cement or asbestos-reinforced plastic for laboratory table tops and electrical and heat insulator applications. Local exhaust provided around the saw blade and the cut will keep fibre levels down to 2 to 4 f/cc if very high rates of airflow are maintained. Another control method for this type of process is wet-cutting, in which a spray of water is continually directed at the cutting area. However, the slurry which results is asbestos-contaminated. If this water dries in floor cracks and in parts of the machinery, then a fibre-contaminated cake is produced. Any disturbance of this cake will result in fibre contamination of the air. Wet-cutting could not produce fibre levels below approximately 0.5 f/cc. If 0.1 f/cc is desired, the only satisfactory procedure would be to provide the operator with personal protective devices.

We do not believe that any manufacturers in Ontario currently *spin* asbestos yarn from raw asbestos fibre. However, some plants do braid or

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[18] Ibid., pp. 55-56.

[19] Ibid., p. 57.

twist yarn from outside the province into a rope-like material for packing bearings. Generally, the yarn is treated with various substances such as graphite and Teflon before braiding, which reduces dust release in the braiding process. The dust level, however, depends upon the stage of manufacturing at which the yarn is coated. Professor Bragg concluded that a major reduction from 2 f/cc to approximately 0.1 f/cc may be achieved in the braiding and twisting operation through the use of good enclosures and exhaust systems. He did not express confidence that a 0.1 f/cc standard could be met all the time in this process, but rather that this level could be approached in most plants with appropriate ventilation and enclosure.

The *sheeting* process consists of mixing some wet and dry ingredients, including asbestos fibres, and then shovelling the resulting mud-like material into a sheeting mill. The sheeting mill rolls the material into a sheet of a desired thickness, after which some of the moisture is squeezed out and the sheet is cured and hardened. Because the recipe generally includes a large quantity of volatile solvents, the process requires a high degree of local exhaust in order to control the vapour from these solvents. This ventilation also controls asbestos fibre levels. Bragg concluded that a major reduction from 2 f/cc to approximately 0.1 f/cc may be achieved with the use of good enclosure and exhaust systems. Again, Bragg was not confident that a 0.1 f/cc control limit could be consistently met, but rather that exposure levels could be brought to approximately 0.1 f/cc most of the time.

In summary, Bragg found that current Ontario fibre levels in manufacturing are low compared to fibre levels of a decade or more in the past. He also found that further reductions in fibre levels are possible for many processes. However, as actual fibre levels are reduced below 1 f/cc, it becomes more difficult to measure them accurately and more difficult to control them reliably. Thus, success in fibre control leads inevitably to problems of measurement and enforcement, which will be discussed in Chapter 7, Section B of this Report.

C.3 Cost of Control: Three Hypothetical Cases

The information available on the cost of controlling asbestos fibre exposure in the workplace comes from some American studies, and the work by Bragg. Since Bragg has reviewed the American studies, and used them as a source of some input data to his own analysis, we will refer primarily to the Bragg study itself. We will review the cost of controlling worker exposures to asbestos in three types of plants that have counterparts in Ontario: brake manufacture, gasket manufacture, and a small plant whose product line includes both asbestos-containing and non-asbestos gaskets. In each case, the plant is hypothetical, but permits the calculation of typical costs of achieving various control limits.

(a) Brake Manufacture

The first costing exercise involves a hypothetical plant manufacturing disc brake pads and/or shoes for drum brakes. Professor Bragg relied upon the experience of the Ontario brake plants in controlling dust to 1980 levels, and on his calculations of the component costs for dust control systems to produce still lower levels of dust.[20] Rather than calculating the cost for an existing brake plant, he used as an example a hypothetical plant employing about 30 production workers, with one mixing machine, and ten machines each in the preform press, drilling, and grinding/finishing stages. By using this hypothetical plant, it is possible to generate a representative cost estimate for the industry without becoming too complex or specific to a particular plant. Table 6.9 shows the control technology needed and the cost of that technology for the four major processes in the hypothetical brake plant, to comply with control limits of 2, 1, 0.5, and 0.2 f/cc.

While costs are presented separately for each process, in some cases this cost allocation was arbitrary, since major costs, such as the cost of the blower and bag-house, were shared among all processes. Table 6.9 shows the capital cost of the equipment for each process, the annual operating cost including labour, materials, and reduced productivity, the number of operators, and the total annual cost per operator. Actual control costs will depend upon the size of the plant and its age, the type of equipment currently installed, the skill and expertise of the operator of the plant, and other factors, so that these estimates cannot necessarily be applied directly to any particular manufacturing plant.

Compliance with a 2 f/cc control limit is estimated to require a $94,920 capital investment and a $100,602 per year operating cost, for an average total cost per operator of $4,083 per year. The data on worker exposure shown above in Table 6.7 suggest that by 1980 and 1981 most brake manufacturing plants exceeded a 2 f/cc level only 10 to 20% of the time. Thus, the industry was close to compliance with a 2 f/cc control limit. Therefore, we may assume that the $4,083 per operator for achieving a 2 f/cc control limit has already been spent. Moving to a 1 f/cc control limit requires a total expenditure of $5,432 per year per operator, an increase of $1,350 per operator per year, and Bragg regarded the 1 f/cc control limit as difficult to meet in the debagging operation. Compliance with a 0.5 f/cc control limit costs $7,954 per year per operator, an increase of more than $2,500 per year per operator over the cost of achieving 1 f/cc. Once again, Bragg was not at all optimistic that this level could in fact be complied with without respirators for debagging.

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[20] Ibid., sec. 4.2.

Finally, the cost estimate for 0.2 f/cc is a total capital cost of $2.125 million, an annual operating cost of $173,502, and an annual cost per operator of $19,721, an increase of almost $12,000 per year per operator over the cost of complying with a 0.5 f/cc control limit. Professor Bragg was not convinced that compliance with the 0.2 f/cc control limit could be achieved in practice throughout most of the plant on a regular basis.

(b) Gasket Manufacture

The second hypothetical costing exercise involves a manufacturer of gaskets and braided packings. The manufacturer is assumed to braid asbestos yarn into packing rope, and to manufacture gasket sheeting and then stamp gaskets out of that sheeting. For purposes of this costing study, a hypothetical plant employing 30 people and consisting of a compounding or mixing station, a sheeting mill, ten braiding/twisting machines, and a spooling station was analyzed. The costs of complying with a 2 fibre control limit and a 0.2 fibre control limit only were estimated. The results are shown in Table 6.10. While Table 6.10 shows the control technology and cost necessary to comply with a 0.2 fibre control limit in a hypothetical plant, Bragg was again not confident that this could be reliably achieved in practice.

One might therefore interpret these costs as the costs of reliably complying with or bettering a control limit of 0.5 f/cc, or as a cost of approaching, if not meeting, compliance with a 0.2 fibre control limit. Interestingly, while most of Bragg's concern about achieving low fibre levels was in the debagging and mixing operation, the major costs shown for complying with the 0.2 fibre limit in Table 6.10 occur at the braiding operation. The cost of approaching the 0.2 fibre control limit, at $6,996 per operator per year, is only about $1,800 greater than the cost of complying with the 2 fibre control limit. Fibre control in this industry is clearly less expensive than in friction products, judged by a comparison of the average total cost per operator per year for approaching a 0.2 fibre level in the two industries.

(c) Small Gasket Manufacture

The third costing exercise is for a small manufacturer of gaskets, whose product line includes both asbestos-containing and non-asbestos gaskets. The plant includes two presses, two hand-cutting tables, and two sewing machines, employing eight production workers. The plant does not handle raw asbestos, but buys sheets of gasket material, so the dirtiest operations, bag-opening and mixing, are avoided. Even in the absence of control limits, fibre levels are in the 0.5 to 0.2 f/cc range. The hand-cutting operation is the only major contributor to airborne asbestos fibres. Professor Bragg concluded that controlling this operation by the use of special surface-exhausted tables can reduce levels to 0.2 f/cc near the cutting table.

Table 6.9

Requirements for Control in Friction Products Manufacturing and Relative Costs
(Costs in 1980 $ Canadian)

Fibre Control Limit	Process	Description of Control	Capital Cost	Annualized Capital Cost	Annual Operating Cost	Number of Operators	Annual Total Cost per Operator*
2 f/cc	Storage, receiving, and mixing	Manual bag opener with local exhaust	$ 12,600	$ 2,362	$ 7,924	2	$5,143
	Preform and press	No local exhaust required	–	–	4,036	9	448
	Drilling	Minimal enclosure design with exhaust	41,160	7,715	44,321	9	5,782
	Grinding and finish	Minimal enclosure design with exhaust	41,160	7,715	44,321	9	5,782
		TOTALS	$ 94,920	$17,792	$100,602	29	AVERAGE $4,083
1 f/cc	Storage, receiving, and mixing	Immediate bag repair; general housekeeping; manual bag opener with good hood or enclosure; increased face velocity	$ 11,025	$ 2,067	$ 8,665	2	$5,366
	Preform and press	Minimal local exhaust	32,393	6,071	28,710	9	3,865
	Drilling	Good enclosure surrounding workplace	47,784	8,967	45,978	9	6,104
	Grinding and finish	Full enclosure	59,202	11,097	45,978	9	6,342
		TOTALS	$150,404	$28,192	$129,331	29	AVERAGE $5,432

Table 6.9 (continued)

Requirements for Control in Friction Products Manufacturing and Relative Costs
(Costs in 1980 $ Canadian)

Fibre Control Limit	Process	Description of Control	Capital Cost	Annualized Capital Cost	Annual Operating Cost	Number of Operators		Annual Total Cost per Operator*
0.5 f/cc	Storage, receiving, and mixing	Immediate bag repair; general housekeeping; tighter enclosures about the entry port and debagger; larger general hood	$ 128,961	$ 24,170	$ 32,098	2		$28,134
	Preform and press	Full local exhaust	44,130	8,273	26,718	9		3,888
	Drilling	Full enclosure	65,926	12,367	57,343	9		7,744
	Grinding and finish	Full enclosure	65,926	12,367	57,343	9		7,744
		TOTALS	$ 304,933	$ 57,157	$173,502	29	AVERAGE	$7,954
0.2 f/cc**	Storage, receiving, and mixing	Covered (protected) storage containers; increased floor space; fully automated debagger and mixing set with large exhaust hood	$ 228,962	$ 42,915	$ 32,098	2		$37,507
	Preform and press	Full enclosure	79,262	14,856	26,718	9		4,619
	Drilling	Full enclosure	141,207	28,468	57,343	9		9,312
	Grinding and finish	Full enclosure and perhaps redesign special equipment for specific finishing operations	1,676,150	314,178	57,343	9		41,280
		TOTALS	$2,125,571	$398,417	$173,502	29	AVERAGE	$19,721

Notes: *Annual Total Cost per Operator equals Annual Capital Cost plus Annual Operating Cost, divided by Number of Operators.
**This level may not be achievable in practice.

SOURCE: Adapted from: Gordon M. Bragg, *The Technical Feasibility and Cost of Controlling Workplace Exposure to Asbestos Fibres*,

Table 6.10

Requirements for Control in Gasket Manufacturing and Relative Costs
(Costs in 1980 $ Canadian)

Fibre Control Limit	Process	Description of Control	Capital Cost	Annualized Capital Cost	Annual Operating Cost	Number of Operators	Annual Total Cost per Operator
2.0 f/cc	Debag, mixing, receiving	Hooded debagging mixing stations	$ 8,143	$ 1,526	$ 6,069	3	$ 2,532
	Sheeting mill	Minimal hood design and flow rate	34,277	6,425	30,241	5	7,333
	Trimming tables	Minimal enclosure	—	—	4,036	6	673
	Braiding M/C	Minimal enclosure	97,802	18,332	56,446	10	7,478
	Spooling M/C	Canopy hood	7,071	1,325	6,069	1	7,394
		TOTALS	$147,293	$27,608	$102,861	25	AVERAGE $ 5,219
0.2 f/cc*	Debag, mixing, receiving	Debagging with enclosure	$ 6,897	$ 1,293	$ 4,552	3	$ 1,948
	Sheeting mill	Large canopy hood at 130 ft/min face velocity	52,837	9,904	45,933	5	11,167
	Trimming tables	Complete (tight) enclosure	21,756	4,078	12,000	6	2,680
	Braiding M/C	Complete (tight) enclosure	120,241	22,538	68,969	10	9,151
	Spooling M/C	Better placement for particle capture; increased cfm	5,825	1,092	4,552	1	5,644
		TOTALS	$207,556	$38,905	$136,006	25	AVERAGE $ 6,906

Note: *This level may not be achievable in practice.

SOURCE: Adapted from: Gordon M. Bragg, *The Technical Feasibility and Cost of Controlling Workplace Exposure to Asbestos Fibres*, Royal Commission on Asbestos Study Series, no. 7 (Toronto: Royal Commission on Asbestos, 1982), Table 4.5, p. 79.

C.4 Economic Impact of Control Costs

What are the economic consequences of the cost increases that may result from the requirement in one province of measures more strict than in other provinces, or in the United States, to protect the health and safety of workers? Any of three results is possible. First, manufacturers may raise prices and pass their increased costs fully on to consumers. Alternatively, manufacturers might pay lower wages to their workers, and thereby pass the costs back to those workers. Finally, prices and wages might remain unchanged, with investors in the industry absorbing all the costs through a lower rate of profit. In many cases, some combination of these three effects will result.

What determines the extent to which each effect occurs? The primary factor is the degree of competition in each market. If the product market prices are determined primarily by competition from firms outside the province, firms in the province have little ability to raise prices without losing markets to their outside competitors. If there is little or no competition from non-Ontario manufacturers for the market served by Ontario manufacturers, they may be able to pass on most or all of their cost increases to consumers. If the labour market is highly competitive, then wages cannot fall without current workers moving to other jobs, and difficulties occurring in recruiting new workers. On the other hand, if workers have few or no alternative places of employment, it may be possible to lower wages sufficiently to recoup the extra costs. Finally, if the shares of the firm are publicly traded, then in the long run the firm must pay its shareholders a market rate of return if it is to attract new capital. If the industry has been earning above-average rates of return, these may be driven down to the average, before difficulty arises in attracting new capital.

We have some information on the competitiveness of the markets for products of Ontario asbestos manufacturing firms. It was suggested earlier in this chapter that most Ontario manufacturers face some competition from other provinces and from the United States. For those plants, it could be assumed that significant price increases would be accompanied by significant losses in market share. If U.S. manufacturers abandon asbestos product lines in the future because of concern about tort lawsuits, then the effective degree of competition will be reduced, and the ability of Ontario manufacturers to raise their prices will be enhanced. Some product lines, however, face competition from other provinces, which might cause substantial erosion of market shares in the event of substantial price increases. If other Canadian provinces adopt regulations similar to those in Ontario, then the relative competitive position of Ontario versus other provinces should not be significantly affected, and passing costs on to consumers would be facilitated.

We have little data on the alternative opportunities facing workers in the asbestos manufacturing firms of Ontario. Frequently, the skill level required of exposed workers is not high, so that they are not prevented from moving to other jobs by possessing skills that are specific to this particular activity. Some plants are located in large labour markets where alternative employment opportunities should exist, at least in good economic times. A few are located in small centres where one might anticipate that even in good times, alternative employment opportunities would be limited. Here, the ability of the firm to reduce wages to absorb cost increases would probably be greater than in plants located in large labour markets. Of course, to the extent that labour perceives more strict asbestos control standards as enhancing the quality of the workplace, then fibre control programmes should increase the supply of labour at any particular wage, or allow firms to lower wages slightly and still attract a sufficient pool of workers.

Most of the asbestos manufacturing firms in Ontario are related to large multinational corporations. Here it seems safe to assume that in the long run, rates of return below the return earned by other segments of the corporation would lead to a reduction or elimination of asbestos manufacturing. We have no way of knowing the extent to which Ontario asbestos manufacturing plants may currently generate above-average rates of return which would allow reduced earnings without reduced investment.

There are two comparisons that may be helpful in assessing the magnitude of control costs. First, we can compare the costs per year per operator per fibre with the wages of the operators themselves. Table 6.3 reports average weekly earnings in asbestos manufacturing in Canada of $322 per week in 1980. If we multiply by 50 work weeks per year, we get an annual wage in 1980 of approximately $16,000. By comparison, the cost of moving from 2 to 1 f/cc in the brake plant is $1,349 per operator per year, representing 8% of the worker's wage. The 0.5 f/cc level costs $3,871 more per year than the 2 f/cc level, or about 24% of the wage. Finally, the 0.2 f/cc level, if it could be achieved, would cost $15,683 per operator per year more than the 2 fibre level, representing about 98% of the worker's wage. Thus, moving from 2 f/cc to 0.2 f/cc would be approximately as costly as doubling the wage rate of those operators who are directly exposed in the manufacturing process.

Suppose that the industry could pass these costs on entirely to their customers. What price increase would be required? We can only make rough estimates of the answer to this question. Table 6.2 shows that in 1980 the Ontario manufacturing industry as represented in that table employed 1,195 production workers. Let us suppose that all of these workers were employed in the friction products industry and that all of them were operators in the sense used by Bragg in his cost calculations. The total cost of control for the industry can be determined by multiplying the cost per year per operator by 1,195 operators. Thus, achieving the 1 f/cc level adds

$1,349 per worker to the firm's cost, for a total of $1.6 million. Since the industry's wage bill, shown in Table 6.2, is $27.3 million, this should be a 5.9% increase in the total cost of employing and protecting these workers. Data from Statistics Canada show the value of asbestos products produced by the industry in 1980 at $66.4 million. If prices were increased to generate an additional $1.6 million of revenue, this would require a 2.4% increase in product prices.

Applying the same analysis to the 0.5 f/cc level yields a control cost of $3,871 per operator per year, which would impose a total cost of $4.63 million to protect 1,195 workers. This would raise the total wage bill by 17%, and would require that product prices be raised by 7% to cover the cost.

While these comparisons may be instructive, they are nevertheless crude. The data in Table 6.2 are from a variety of operations, not just brake plants, and other operations may be controlled at less cost than brake plants. Furthermore, not all hourly employees would be operators as defined by Bragg, and therefore the cost of controls for the industry may be considerably less than the estimates just presented. If half of the production employees were not operators, but were performing maintenance and other duties, then the industry costs we have just calculated would be overstated by a factor of 2. This analysis of control costs relies upon existing technology, which Bragg expects to be the predominant technology for the immediate future. It is entirely possible that in the future, advanced technology might reduce the costs below those found here. Automation might greatly reduce the number of exposed workers and in consequence reduce the cost of worker protection; however, it might also generate adverse employment effects. Finally, all costs will not necessarily be passed on to consumers. It is possible that some costs would be absorbed by labour in trading lower wages for better health and safety, and some might be absorbed out of the profit of the firms, if this still allows an adequate rate of return.

C.5 Marginal Cost of Control per Marginal Unit of Exposure

Table 6.9 above presents detailed information on the costs of controlling fibre levels in a hypothetical brake manufacturing plant. These data are summarized in the annual cost per operator of achieving the various fibre levels. The calculations can be carried one step further to determine the cost per fibre per cubic centimetre of reducing operator exposure. This is the difference between the cost for any two fibre levels divided by the fibre reduction. If it costs $1,000 to reduce exposure from 1 f/cc to 0.5 f/cc, the marginal cost per fibre would be $1,000 divided by a 0.5 fibre reduction or $2,000/f/cc. If it costs another $1,000 to reduce the exposure from 0.5 f/cc to 0.2 f/cc, the marginal cost per fibre would be $1,000 divided by a 0.3 fibre reduction or $3,333/f/cc. The same cost increment yields a marginal cost per fibre that is two-thirds greater because it achieves a fibre reduction only 60% as great.

The initial achievement of compliance with a 2 f/cc control limit (1 f/cc exposure) costs $4,083 per year per operator. While we do not know what levels would exist in the absence of controls, Berry and Newhouse estimated that average fibre exposures in the Ferodo friction products plant at Derbyshire, England, were often around 5 or 10 f/cc.[21] Assuming that uncontrolled levels in Ontario brake plants could be as high as 10 f/cc, then the reduction of exposure from 10 to 1 f/cc would cost $454 per operator per year per f/cc. In the case of the data presented in Table 6.8, reducing the control limit from 2 to 1 f/cc (1 to 0.5 f/cc exposure) costs $1,349 per worker per year for a reduction of 0.5 f/cc, for a marginal cost of $2,700 per year per operator per fibre. Reducing the control limit from 1 to 0.5 f/cc (0.5 to 0.25 f/cc exposure) costs $2,522 per year per operator. Dividing this by the reduction in fibre level of 0.25 fibres yields a cost per year per operator per fibre of $10,088. Similarly, reducing the control limit from 0.5 to 0.2 f/cc (0.25 to 0.1 f/cc exposure) costs $78,447 per year per operator per f/cc. Figure 6.1 graphs the marginal cost of moving fibre control to the various levels just discussed. While the calculations have produced just four data points, we have drawn a smooth curve through these points in Figure 6.1. This implies that fibre levels *between* those analyzed could be achieved at the marginal cost indicated by the figure. The band on either side of the figure represents the range of uncertainty surrounding these cost estimates.

We conclude from this exercise and Figure 6.1 that the cost of controlling workplace exposure to asbestos may rise rapidly as control levels are reduced to and below the current Ontario control limit for chrysotile. Plausible control levels can yield costs of worker protection that are as great as the salary of the worker. Choices about control limits in this range can therefore substantially affect the cost of asbestos manufacturing in Ontario. We also conclude that while there are many sources of error and uncertainty in making such cost estimates, there is still substantial value in making them.

We now turn to another means of worker protection: the substitution of other materials for asbestos products.

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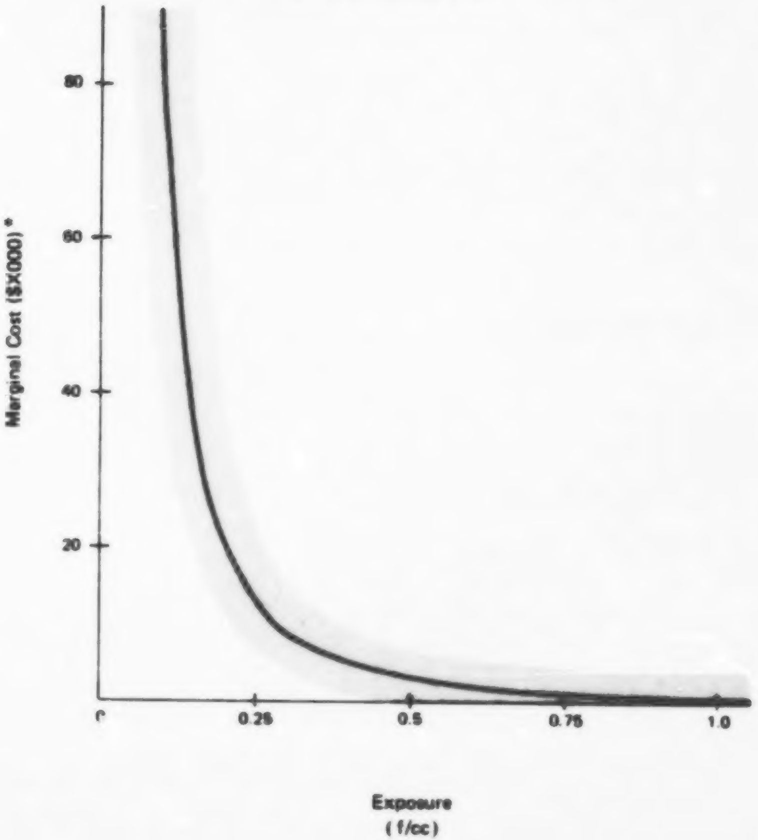
[21] Geoffrey Berry and Muriel L. Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," *British Journal of Industrial Medicine* 40:1 (February 1983): 1-7.

Figure 6.1

Marginal Cost of Fibre Control
Hypothetical Brake Plant



Data:	Control Limit (f/cc)	Exposure (f/cc)	Marginal Cost*
	2.0	1.0	484
	1.0	0.5	2,700
	0.5	0.25	10,088
	0.2	0.1	78,447

Note: *Marginal total cost per year per operator per fibre of exposure.

D. Feasibility and Cost of Substitutes for Asbestos

The exposure of manufacturing workers to asbestos can be eliminated by replacing asbestos in the product with some other fibre, or by replacing the product with a non-fibrous product. Considerable work has been done particularly in the United States during the last few years to identify and develop substitutes for asbestos. This search has found a variety of materials that can substitute for asbestos in various applications, but no single material that is satisfactory for all of the varied uses of asbestos. Thus, an investigation of asbestos substitutes must be conducted separately for each product type. We will review briefly here the available information on substitutes for the major products manufactured in Ontario.

D.1 Friction Products

Friction materials are used in brakes and clutches for automobiles, trucks, airplanes, railroads, and industrial machinery. In the past, asbestos has been a major component of the pads used in disc brakes and in the linings of drum brakes. Substitutes for asbestos must be able to withstand high temperatures, maintain strength and durability, provide uniform friction characteristics over a wide range of temperatures and under wet and dry conditions, and not cause unreasonable wear for the brake disc or drum. Substitute materials include semi-metallic friction materials, cermets (ceramic and metal mixtures), as well as glass fibres, steel wool, mineral wool, carbon fibres, aramid fibres, vermiculite, silicon nitride, and potassium titinate fibres. Jacko, Brunhofer, and Aldrich reported that semi-metallic materials have been used in disc brake pads for special applications for a number of years, and are now being introduced in automobiles.[22] In 1980, semi-metallic disc pads were used on the front brakes of approximately half of all new North American vehicles, and it is expected that this fraction will approach 100% by 1985. Semi-metallic disc pads are somewhat more expensive than asbestos pads, but their performance is sufficiently good that this cost disadvantage has not prevented widespread acceptance. The GCA Corporation, in a report drafted for the U.S.

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[22] Michael G. Jacko, Charles M. Brunhofer, and F. William Aldrich, "Non-Asbestos Friction Materials," in *Proceedings of the National Workshop on Substitutes for Asbestos, Arlington, Virginia: 14–16 July 1980*, EPA-560/3-80-001 (Washington, D.C.: U.S. Environmental Protection Agency, 1980), p. 9.

Environmental Protection Agency, stated that most commercial airliners have used cermet disc brake pads for decades.[23]

Until recently, all drum brake linings were still asbestos-based. In drum brakes, the semi-metallic materials are not appropriate, and research is focused upon the alternative fibres mentioned above. Some of these fibres are difficult to handle in processing because they are brittle or difficult to spread uniformly throughout the mixture of ingredients prior to forming. In general, they have shown less frictional stability, greater noise problems, and excessive wear on the brake drum. However, research is continuing, and in the summer of 1983 two manufacturers had introduced non-asbestos drum brake linings on a limited scale.[24]

The cost of alternate brake shoe materials is difficult to predict. Jacko, Brunhofer, and Aldrich reported that substitute fibres alone range in cost from slightly cheaper than asbestos to 99 times more expensive than asbestos.[25] They estimate that asbestos-free drum brake linings may cost 20 to 50% more than current linings, while asbestos-free disc pads may cost 20 to 100% more than current pads.[26] The GCA Corporation report anticipated similar cost increases, except that they predicted a cost increase for drum brake linings of 20 to 25% above the cost of current linings.[27]

Even if all new vehicles were manufactured with non-asbestos disc and drum brake linings, existing vehicles would continue in use for many years. It is generally believed that replacement parts for the existing vehicles will have to be of a composition similar to that of the original product in order to maintain adequate performance. Thus, there may be a continuing demand for asbestos-containing brake shoes for the replacement market for many years after asbestos-free linings are developed.

A survey of Ontario manufacturers conducted by Professor Bragg as part of his study for this Commission showed that some firms in the friction products industry intended to phase out asbestos within five to ten years, while others had no plan to phase out asbestos. It appears that one firm has been given the responsibility for manufacturing all asbestos brake materials for the parent company in North America, so that production has been shifted from the U.S. plant to this Ontario plant.

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[23] GCA Corporation, "Asbestos Substitute Performance Analysis," draft revised final report prepared by Nancy Krusell and David Cogley for the U.S. Environmental Protection Agency, GCA-TR-81-32-G (Bedford, Mass.: GCA Corporation, February 1982), p. 312. The information from the GCA Corporation report is currently being incorporated by the U.S. Environmental Protection Agency into an updated report, tentatively titled "Regulatory Impact Analysis for Asbestos Product Bans," in press, 1983. See especially Appendix B. Telephone communication between Ms. Amy Moll, Office of Toxic Substances, U.S. Environmental Protection Agency and Royal Commission on Asbestos Staff, 13 September 1983.

[24] These manufacturers are Raybestos-Manhattan and the BBA Group.

[25] Jacko, Brunhofer, and Aldrich, "Non-Asbestos Friction Materials," p. 17.

[26] Ibid., p. 24.

[27] GCA Corporation, "Asbestos Substitute Performance Analysis," p. 104.

D.2 Floor Tiles

Asbestos has long been used in vinyl-asbestos floor tiles, because it gives durability, dimensional stability, moisture resistance, a smooth surface, and easy cleaning. In 1975, asbestos-containing products commanded a 91% share of the resilient floor market.[28] Some companies are testing alternate fibres to substitute for asbestos in floor tiles. This research is generally proprietary, so little is known about the characteristics of floorings containing these alternate fibres. There are also alternative products including solid vinyl tiles, rubber tiles, and linoleum. Each of these products is said to be inferior to vinyl-asbestos tile in some important respects such as grease resistance, stain resistance, or durability. The substitutes tend to be considerably more expensive than vinyl-asbestos floor tile, with price premiums of 10 to 50%.[29] Thus, at the present time, there are alternatives to vinyl-asbestos floor tile, but they do not have the cost and performance characteristics of the vinyl-asbestos product. One Ontario manufacturer has placed on the market an asbestos-free vinyl floor tile, and while its cost and market acceptance are not as yet known, there appears to be some demand for an asbestos-free product.

D.3 Gaskets and Packings

Asbestos is used in gaskets and packings to provide heat resistance, resiliency, strength, and resistance to strong chemicals. A number of fibres have been tested to replace asbestos in gaskets and packings, including graphite, carbon, aramid, cermet, silica, and Teflon fibres. Some non-fibrous gasket materials also exist. Aramid fibres are strong and low cost, providing a long surface life, but are hard to cut and have a 500°F temperature limit. Graphite/TFE compositions are also attractive for packing, withstanding a wide range of acid or alkali conditions, with a number of attractive characteristics, particularly for packing chemical pumps. Carbon fibre is expensive, but can withstand high shaft speeds and high temperatures. In the gasket product, a variety of fibre and non-fibre alternatives exist, which when combined cover virtually the entire range of asbestos product applications.

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[28] Ibid., p. 173.

[29] Ibid., p. 179.

The GCA Corporation report concluded that "New packing materials appear to be more than viable alternatives [to asbestos], offering less abrasion and thus lower operating and maintenance costs. It appears that only sales and engineering resistance stand in the way of a total switchover to non-asbestos packings."[30] The U.S. Environmental Protection Agency's *Proceedings of the National Workshop on Substitutes for Asbestos* also appeared to conclude that the variety of asbestos substitutes left few if any users locked into asbestos products for gaskets and packings. The cost penalty for a non-asbestos product is thought to be negligible except in high temperature applications. However, Pye has noted that asbestos can cover a wide range of uses, while a variety of substitutes might be needed to cover the same range.[31] Thus, the continued use of asbestos gaskets and packings can help to minimize inventory problems.

D.4 Paints, Sealants, and Coatings

Asbestos is used in paints, sealants, and coatings such as automotive undercoating and asphalt roofing compounds, because it absorbs sound, is waterproof, adds strength, reduces flow or sag of the coating, and is durable, economical, and resistant to corrosion and heat. Asbestos mixes and binds well with petroleum products and particularly asphalt. Alternative substitute fibres do not in general have this affinity for asphalt, which give them less satisfactory performance. Thus, a number of asbestos-free substitutes are available, but they tend to be more expensive and less durable or otherwise less satisfactory than asbestos-containing coatings. Brzozowski reported that the cost of asbestos substitutes for roof coatings are from 2 to 20 times greater than the cost of the asbestos itself, which means that the finished product is 10 to 150% more expensive than the asbestos-containing product.[32] The non-asbestos fibres that have been used in roof coatings are generally not inert, so that over time they degrade or settle or are attacked by chemicals. Thus, the asbestos-free products provide lower durability and performance than the asbestos-containing products.

Textured paint, spackling, and drywall joint compounds used to contain asbestos, but now are generally asbestos-free. For non-asphalt coatings, a wide variety of asbestos substitutes have been used including talc, silica, clay, and mica. For pipe coatings, there are a number of asbestos-free alternatives including enamels, plastics, phenolic tapes and wax coatings, polyurethane foam insulation, and concrete. The problems with these substitutes are substantially less than with the asphalt products. Ontario manufacturers of paints, sealants, and coatings indicated that some had abandoned asbestos use, while others were investigating asbestos substitutes.

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[30] Ibid., p. 314.

[31] Andrew M. Pye, "The Feasibility of Substitution," in *Proceedings of the World Symposium on Asbestos*, Montreal, Quebec: 25–27 May 1982 (Montreal, P.Q.: Canadian Asbestos Information Centre [1983]), p. 216.

[32] Kenneth Brzozowski, "Asbestos Substitutes in Roof Coatings," in *Proceedings of the National Workshop on Substitutes for Asbestos*, p. 109.

D.5 Plastics

Asbestos has been used as a filler to provide strength in phenolic molding compounds and other plastic products. Here asbestos provides a filler and reinforces the plastic, giving it strength, resistance to heat and fire, and less shrinkage and warping than other fibres. A variety of asbestos substitutes are available for this use including clay, talc, wollastonite, glass fibres and processed mineral fibres. The GCA Corporation report concluded that most of these substitutes are economically competitive with asbestos and yield satisfactory product qualities.[33] Some of the substitutes, however, cost many times as much as asbestos fibres, and the resulting product is more costly to process.[34] There has been a move from asbestos to these substitutes in recent years, a trend which is expected to continue, leaving asbestos only in some specialty products. An Ontario manufacturer of reinforced plastics, however, reported that it felt non-asbestos substitute products to be inferior and had no plans to phase out asbestos within the next decade.

D.6 Asbestos-Cement Pipes and Sheets

At present there is no manufacturer of asbestos-cement pipe in Ontario, but the Johns-Manville plant in Scarborough was the major asbestos user in the province from the late 1940s until 1980. Asbestos-cement pipe has been used for sewer pipe and for water supply piping. The asbestos reinforces the cement mixture, giving strength to the pipe, as well as providing resistance to corrosive soils. Several alternative fibres are under consideration for pipe manufacture, including fibreglass, while iron pipe and plastic pipe are competing products for water supply, and clay pipe and concrete pipe are competing products for sewage and drainage pipes. Thus, it appears for most applications that at least one alternative to asbestos-cement pipe will offer satisfactory performance, and the main factor of choice is economics. Asbestos-cement pipe may cost 25% less than ductile iron pipe for diameters up to 12 inches, and it is easier to tap.[35] It appears that for small pressure pipes, plastic has a competitive edge over asbestos, in part because it is light-weight and requires less field labour to install. The GCA Corporation report concluded that asbestos-cement pipe is still most suitable for diameters of 6 to 24 inches, although others have suggested that plastic pipe is highly competitive over some of this size range.[36]

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[33] GCA Corporation, "Asbestos Substitute Performance Analysis," p. 315.

[34] Ibid., pp. 258–259.

[35] Ibid., pp. 142–143.

Bragg reported that very little asbestos-cement pipe seems to be installed in Ontario today. He concluded that the major Canadian market for this pipe is in the western provinces, with plastic and other types of pipe having taken over most of the eastern market.[37]

E. Health Effects of Substitutes for Asbestos

The preceding section identified competitive materials which could serve as substitutes for asbestos in various product applications. However, in addition to performance and economic criteria, one must incorporate consideration of the health effects of alternative materials into the evaluation of substitutes. It would be unfortunate if asbestos were replaced with a substance characterized by greater carcinogenic or fibrogenic properties.

Many problems complicate the appraisal of the health consequences of alternative materials, including: (i) diversity and definition of asbestos substitutes; (ii) the incomplete understanding of the toxicology and pharmacokinetics of fibre particulates; (iii) the limited development of scientific criteria for determining toxicities due to factors (i) and (ii); and (iv) the uncertainty involved in the assessment of quantitative risks to humans by extrapolation from toxicological data in other species.[38]

Here we will summarize the results of epidemiological and animal studies purporting to assess the health effects of substitute materials for asbestos. The available substitutes for asbestos are classified into three broad categories: synthetic fibre substitutes; natural fibre substitutes; and natural non-fibre substitutes.

E.1 Synthetic Fibre Substitutes

Man-made vitreous fibres (MMV) such as fibrous glass, rock and slag wool, and ceramic fibre may be used as substitutes for asbestos in various insulation applications. Fowler has reported that production workers exposed to man-made vitreous fibres generally experience relatively low exposures to the material (less than 1 f/cc), while user workers have more variable exposure.[39]

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[36] Ibid., p. 313.

[37] Bragg, *The Technical Feasibility and Cost of Controlling Workplace Exposure to Asbestos Fibres*, p. 85.

[38] James N. Rowe, "Scope of the Health Workshop," in *Proceedings of the National Workshop on Substitutes for Asbestos*, p. 279.

Konzen, after surveying the available medical literature on man-made vitreous fibres, concluded that in man ". . . there is no credible evidence of malignant or chronic progressive non-malignant disease resulting from exposure to man-made vitreous fibers." In laboratory animals, disease was produced only ". . . when fibers were administered by an artificial route which bypassed physiologic defenses."[40] And, although a recently completed epidemiological study by Robinson et al. detected increased mortality related to gastrointestinal cancer and non-malignant respiratory disease in workers previously employed in a rock and slag wool factory, the excess was not considered statistically significant at the 95% confidence level.[41] As of 1980, when Konzen and Robinson reported, this leaves some possibility of adverse health effects, but not *proven* health effects.

Recently, Enterline, Marsh, and Esmen studied the health of workers exposed to fibreglass and mineral wool.[42] After 20 years from first exposure, they found standard mortality ratios (SMRs) for respiratory cancer of 108 among the fibreglass workers and 157 among the mineral wool workers. The estimated exposure to respirable dust was 0.5 f/cc for fibreglass and 5 f/cc for mineral wool, the former being a very low exposure level. While the fibreglass disease rate was very low, so was the exposure. If one develops a dose-response relationship from the data contained in this study, the cancer risk per f/cc of exposure is in fact relatively high. Finkelstein concluded that these data imply an increase in relative risk of 16% per f/cc-yr and 12% per f/cc-yr for fibreglass and mineral wool exposure respectively.[43] These risks are twice as great as the cancer risks from asbestos exposure found at the Johns-Manville plant in Scarborough. This study suggested that the failure to detect high disease levels among fibreglass or mineral wool workers may arise from the low concentrations of respirable dust to which they were exposed. If changes in the manufacturing processes for these materials were to lead to airborne concentrations of respirable fibres substantially above the low historical levels, serious disease might result. Specifically, risks might increase if there were a reduction in the diameter of fibres of these types that are produced, and thus to which workers are exposed. A 1977 criteria document by the U.S. National Institute for Occupational Safety and Health (NIOSH) recommended that worker exposure to fibreglass in the United States be limited to 3 f/cc of fibres thinner than 3.5 microns and longer than 10 microns.[44]

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[39] Douglas P. Fowler, "Occupational Exposure to Mineral Wool," in *Proceedings of the National Workshop on Substitutes for Asbestos*, p. 412.

[40] Jon L. Konzen, "Man-Made Vitreous Fibers and Health," in *Proceedings of the National Workshop on Substitutes for Asbestos*, pp. 329, 332.

[41] Cynthia F. Robinson et al., "Mortality Patterns of Rock and Slag Mineral Wool Production Workers: Epidemiologic and Environmental Study," in *Proceedings of the National Workshop on Substitutes for Asbestos*, p. 419.

[42] Philip E. Enterline, Gary M. Marsh, and Nurtan A. Esmen, "Respiratory Disease Among Workers Exposed to Man-Made Mineral Fibers," *American Review of Respiratory Disease* 128:1 (July 1983): 1–7.

[43] Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983, p. 20. (Mimeographed.)

E.2 Natural Fibre Substitutes

Boehlecke et al. completed a clinical examination of 104 workers in a wollastonite (a fibrous mono-calcium silicate) plant in the United States and found that 23% of the smokers and ex-smokers and 9% of the non-smokers suffered from chronic bronchitis. However, there was no association between prevalence of bronchitis and increases in exposure to the material. The researchers concluded by cautioning that "Because only 36% of men studied had over 15 years' exposure, further follow-up is needed to determine if wollastonite is truly less hazardous than asbestos."[45]

Rohl has investigated the high mesothelioma mortality in an agricultural region in Turkey.[46] Rohl was unable to detect any evidence of asbestos in the area, and postulated that the carcinogen responsible for inducing the disease was zeolite, a fibrous substance having similar dimensions to some asbestos fibres. This supposition was consistent with the results of animal studies undertaken by Wagner which found that all animals inoculated with zeolite and surviving one week after the procedure eventually developed mesothelioma.[47]

Flowers has proposed that it may be possible to reduce the toxic potency of asbestos fibres while retaining the desirable attributes of the material. This is accomplished by attaching ferric oxides to the fibres.[48] Initial results from *in vivo* testing indicated a reduction in the cytotoxicity of these treated forms of asbestos. However, biological activity was measured from only 72 hours after treatment, so further investigation is needed on the carcinogenic and fibrogenic potential of these treated substances before their relative health effects can be conclusively determined. In testimony before this Commission, Dr. Jacques Dunnigan described the experimental treatment of asbestos fibres with heat, acid, and other compounds, especially phosphate, all designed to reduce the health risk from asbestos fibres while retaining the desirable properties of the fibres.[49] Concomitantly, there has been research on the adverse health effects of these modified fibres. While it is too early to judge the economic feasibility of these treatments, or the reduction they may achieve in health effects, it is possible that in the future a treated asbestos will emerge as a safer substitute for untreated asbestos.

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[44] U.S., Department of Health, Education and Welfare, National Institute for Occupational Safety and Health, *NIOSH Criteria for a Recommended Standard . . . Occupational Exposure to Fibrous Glass* (Washington, D.C.: U.S. Government Printing Office, April 1977), p. 7.

[45] Brian Boehlecke et al., "Cross-Sectional Medical Study of Wollastonite Workers," in *Proceedings of the National Workshop on Substitutes for Asbestos*, p. 475.

[46] Arthur N. Rohl, "Endemic Pleural Disease in Relation to Zeolite Exposure," in *Proceedings of the National Workshop on Substitutes for Asbestos*, p. 477.

[47] J. Christopher Wagner, "Health Hazards of Substitutes," in *Proceedings of the World Symposium on Asbestos*, pp. 249–251, 263–265.

[48] Earl S. Flowers, "Chemical Detoxification of Asbestos Fibers," in *Proceedings of the National Workshop on Substitutes for Asbestos*, p. 489.

E.3 Natural Non-Fibre Substitutes

Talc has been suggested as a substitute for asbestos in some applications. Assessing the health effects of talc is difficult because the mineral is frequently contaminated with other substances, sometimes asbestos. Studies of miners and millers exposed to talc contaminated with amphibole asbestos found some excess in respiratory cancer as well as other respiratory disease.[50] Gamble et al. attempted to correct for the problem of talc contaminated with other minerals with proven fibrogenic and carcinogenic properties by examining workers from three different talc mines in the United States.[51] Each mine was characterized by varying grades of talc purity. The researchers found that there was neither an increase in symptoms of pneumoconiosis nor biologically significant reductions in lung functions among workers from the three mines examined. Yet the amount of bilateral pleural thickening in talc workers was significantly elevated over the levels found in a comparison population not exposed to the mineral.

It has been reported that exposure to other natural non-fibrous substitutes for asbestos such as mica and vermiculite can cause increased prevalence of pulmonary abnormalities in employees processing these materials.[52]

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[49] Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Evidence of Dr. Jacques Dunnigan, 20 August 1981, Volume no. 28, p. 7.

[50] David P. Brown, John M. Dement, and Joseph K. Wagoner, "Mortality Patterns Among Miners and Millers Occupationally Exposed to Asbestiform Talc," in *Dusts and Disease*, eds. Richard A. Lemen and John M. Dement (Park Forest South, Illinois: Pathatox Publishers Inc., 1979), p. 317; Morris Kleinfeld et al., "Mortality Among Talc Miners and Millers in New York State," *Archives of Environmental Health* 14 (1967): 663; and Morris Kleinfeld, Jacqueline Messite, and Mahfouz H. Zaki, "Mortality Experiences Among Talc Workers: A Follow-up Study," *Journal of Occupational Medicine* 16 (1974): 345.

[51] John Gamble, Alice Greife, and John Hancock, "Cross-Sectional Epidemiologic and Industrial Hygiene Survey of Talc Workers Mining Ore from Montana, Texas, and North Carolina," in *Proceedings of the National Workshop on Substitutes for Asbestos*, p. 570.

[52] James Lockey and Stuart M. Brooks, "Health Effects of Vermiculite," in *Proceedings of the National Workshop on Substitutes for Asbestos*, p. 555.

E.4 Assessment

We conclude from this evidence that the widely discussed substitutes for asbestos have not in general been proved to be carcinogenic or toxic. However, most of these substitutes have been used in ways that have not created airborne concentrations of respirable fibres comparable to the asbestos fibre levels of twenty or more years ago. It may be that they are not as hazardous, fibre for fibre, as asbestos has proved to be. However, the evidence does not rule out the likelihood that some of these materials will ultimately be determined to cause cancer or respiratory disease if substantial quantities are inhaled. There are few cases where a substitute is available that has been "proved safe," in part because such proof is very difficult to establish.

In Chapter 5 we conclude that the health hazards caused by exposure to asbestos fibres depend very much upon the dimensions of the fibres, and that long, thin, respirable fibres are of primary concern. This leads us to be extremely cautious about concluding from the evidence discussed above that substitute fibres will not present health hazards in the future. If the production of substitute fibres in the future should increase the exposure of workers to long, thin, durable fibres of dimensions similar to those we have found hazardous for asbestos, we cannot be sure that serious health consequences would not result. In the face of the existing evidence, we believe it would be risky to allow the exposure of workers to respirable fibres longer than 5 microns, with small diameters, of any material, if those fibres are likely to be very durable in the lungs.

F. Mining and Milling in Ontario

Mining of asbestos in Ontario has never contributed substantially to total Canadian output of asbestos and has not been a major component of the Ontario mining industry. Four mines have produced asbestos in Ontario in the recent past. The Munro Mine outside Timmins, Ontario, closed in 1968. No production figures are available, but the Ontario Federation of Labour (OFL) estimated that the total workforce over the life of this mine included 2,000 workers.[53] The Reeves Mine, also near Timmins, was owned by Johns-Manville Canada and closed in early 1975. The Ontario Federation of Labour estimated that this mine employed between 800 and 1,000 workers during its operating life.[54] The OFL stated that between 1969 and 1975, the fibre counts in the Reeves Mine rose from around 2 f/cc in the early months of 1969 to average between 7 and 10 f/cc in 1970. In August

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[53] Ontario Federation of Labour, Written submission to the Royal Commission on Asbestos, #35, January 1981, p. 48.

[54] Ibid.

1972, 4 out of 15 dust counts exceeded 5 f/cc, with a maximum of 22 f/cc. In 1974, the average of all readings is alleged to have been 14.7 f/cc, with individual readings many times this level, and 34 of 54 sampling stations above 5 f/cc. The mine was closed when Johns-Manville concluded that it could not economically meet a 2 f/cc time-weighted average (TWA) exposure limit. The ore body was estimated to have a three-year supply remaining.

The Matachewan Mine in Midlothian Township outside of Kirkland Lake operated between 1975 and 1977, employing a total workforce over this period of approximately 500 workers according to the Ontario Federation of Labour.[55] This mine was owned by United Asbestos, and was the only Ontario asbestos mine identified by Vagt.[56] He stated that the mill had a capacity of 3,600 tons per day of ore and an annual capacity of 100,000 tons of fibre. During the short operating life of this mine, continual problems occurred in trying to achieve the 2 f/cc TWA exposure limit.

The principal mine that has recently operated in Ontario is the Hedman Mine near Matheson, which was not listed by Vagt as an asbestos producer. The mill, located in the town of Matheson, has a capacity of 300 tons per day of ore. The mine and mill employ under 20 workers. The product contains about 20% chrysotile fibre. Another mine near Sharbot Lake currently produces a non-asbestiform tremolite.[57] The Ontario Ministry of Natural Resources recently completed a study reviewing asbestos deposits in the Kirkland Lake - Timmins area, concluding that the potential for commercial deposits of asbestos and talc is good.[58]

Asbestos exposure might also occur in other mines where asbestos fibres are present in the rock. A talc mine operated by Canadian Talc Limited, near Madoc, has shown some asbestos fibre exposure in Ministry of Labour inspections. The Ministry of Labour conducted its air sampling at the rock crushers of three nickel mines in 1981 and found asbestos fibre levels predominantly below 0.1 f/cc with a maximum of 0.22 f/cc.[59]

Table 6.11 shows asbestos fibre exposures in Ontario asbestos and talc mines since 1975. The table shows high exposures occurring in the mid-1970s while average exposures by 1980 were generally within the 2 f/cc guideline.

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[55] Ibid., p. 49.

[56] Vagt, *Asbestos*, p. 6.

[57] Letter from Mr. Dennis Yakota, Ram Petroleum Limited to the Royal Commission on Asbestos, 15 April 1983.

[58] Ulrich Kretschmar and Dianne Kretschmar, *Talc, Magnesite, and Asbestos Deposits in the Kirkland Lake - Timmins Area, Districts of Timiskaming and Cochrane*, Ontario Geological Survey Open File Report 5391 (Toronto: Ontario Ministry of Natural Resources, 1982), p. 125.

[59] Letter from Mr. Hugh M. Nelson, Special Assistant, Industrial Hygiene, Occupational Health and Safety Division, Ontario Ministry of Labour to the Royal Commission on Asbestos, 14 September 1981.

Bragg noted that in open-pit mining, workers may be protected from fibre levels in excess of 2 f/cc by enclosing the work stations on the equipment that they operate.[60] He concluded that ". . . with the exception of blasting crews, enclosures costing approximately $5,000 to $10,000 per operating station would provide fibre levels at 0.1 f/cc except while entering and leaving the working area. It is expected that blasters would have to wear personal protective devices in the presence of high fibre levels." In the milling operation, it is unlikely that fibre levels can be reliably reduced below 2 f/cc. Even this level would require careful maintenance. Bragg concluded that "It is our estimation that only personal protective devices could provide fibre levels below 2 f/cc for personnel in present asbestos mills."[61] While wet-milling processes have been developed and are used elsewhere in the world, water may render the fibre unfit for the uses to which Canadian fibre is usually put. Research is ongoing in Australia trying to find ways to make this process compatible with the major uses of chrysotile asbestos.

G. Other Fixed Place Exposure

In addition to mining and manufacturing, Ontario fixed place workers may be exposed to asbestos in brake repair, in shipbuilding and repair, and in railroad equipment maintenance. The exposures that occur in these activities will be reviewed below.

G.1 Asbestos Exposure During Brake Repair

Chrysotile asbestos is widely used as a major component of automotive brakes, in both disc pads and drum linings. Depending on the type of brake, the asbestos content of the pad or lining will constitute from 25 to 65% of the total product weight. Within the last few years, cost- and performance-competitive non-asbestos substitutes have become available for disc brakes. However, while some of the major brake manufacturers have recently begun to market non-asbestos drum brake linings, they are available only in a limited number of applications. This suggests that we cannot, in the immediate future, rely on the substitution of non-asbestos materials for asbestos in brakes as the sole method of controlling the asbestos exposure received by brake mechanics, but instead must assume that many brakes will continue to contain asbestos.

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[60] Bragg, *The Technical Feasibility and Cost of Controlling Workplace Exposure to Asbestos Fibres*, pp. 84–85.

[61] Ibid., p. 85.

Table 6.11

Summary of Asbestos Dust Levels in Ontario Asbestos and Talc Mines (Fibres per Cubic Centimetre)

Year	Average		Range	% Greater than 2 f/cc
1975	Area	1.4	to 2.4	18
	Personal	3.8	to 10.1	60
1976	Area	2.9	to 45.0	37
	Personal	3.2	to 13.4	63
1977	Area	3.3	to 10.3	58
	Personal	4.9	to 16.2	83
1978	Area	1.3	to 7.2	12
	Personal	2.0	to 6.2	46
1979	Area	0.8	to 1.0	0
	Personal	2.4	to 6.3	NA
1980	Area	2.3	to 6.5	63
	Personal	1.5	to 9.2	14

SOURCE: Ontario Ministry of Labour data.

Automotive mechanics may be exposed to brake dust during inspection, modification, removal, or installation of brake linings. However, the extent of occupational exposure to asbestos in the servicing of disc brakes is considerably less than in the servicing of drum brakes because disc brakes retain little brake wear debris and disc pads seldom require manual modification to ensure a good fit. During the inspection and installation of drum brake linings, the exposure to asbestos may result from the removal of brake wear debris from the drum and back plates. Historically, brake wear debris was most frequently removed by directing compressed air into the brake housing, to blow out the debris deposited in the drum.[62]

A substantial body of scientific literature has reported that the asbestos undergoes significant chemical and physical alteration during braking. While it is generally agreed that the high temperature experienced during braking alters the asbestos, there is still debate surrounding both the extent of this alteration and the identity and characteristics of the wear debris that is produced. For example, Jacko and DuCharme suggested that the high temperature experienced during vehicle braking caused 99.7% of the asbestos in the brake linings to be converted into inert, harmless particles.[63] Similar results were reported by Lynch; Hatch; Hickish and Knight; Anderson et al.; and Williams and Muhlbaier.[64] And, while Alste, Watson, and Bagg concluded that the major effect of braking appears to be the separation of bundles of fibres and reduction of average length without any crystal structure alteration,[65] Jacko has suggested that the wear debris collected by Alste, Watson, and Bagg may have resulted from studying brake linings with poor wear characteristics.[66]

Williams and Muhlbaier conducted 23 analyses of wear products from disc brakes and 24 from drum brakes.[67] They found that the average asbestos content of the wear products was 0.029%, which means that over 99.9% of the asbestos in the brake pad or shoe was transformed chemically or physically so that it was no longer an identifiable asbestos fibre. Furthermore, they found that less than 1% of all asbestos fibres observed were longer than 5 microns.

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[62] Barry Castleman et al., "The Hazards of Asbestos for Brake Mechanics," *Public Health Reports* 90:3 (May-June 1975): 254-256.

[63] Michael G. Jacko and Robert T. DuCharme, "Brake Emissions: Emission Measurements from Brake and Clutch Linings from Selected Mobile Sources," EPA Report 68-04-0020, NTIS PB-222-372 (Southfield, Michigan: Bendix Research Labs, March 1973).

[64] Jeremiah R. Lynch, "Brake Lining Decomposition Products," *Journal of the Air Pollution Control Association* 18 (1968): 824-826; D. Hatch, "Possible Alternatives to Asbestos as a Friction Material," *Annals of Occupational Hygiene* 13:1 (January 1970): 25-29; D.E. Hickish and K.L. Knight, "Exposure to Asbestos During Brake Maintenance," *Annals of Occupational Hygiene* 13:1 (January 1970): 17-21; A.E. Anderson et al., "Asbestos Emissions from Brake Dynamometer Tests," SAE Paper 730549 presented at Society of Automotive Engineers Meeting, Detroit, Michigan, 14-16 May 1973; and Ronald L. Williams and Jean L. Muhlbaier, "Asbestos Brake Emissions," *Environmental Research* 29 (1982): 70-82.

[65] J. Alste, D. Watson, and J. Bagg, "Airborne Asbestos in the Vicinity of a Freeway," *Atmospheric Environment* 10:8 (1976): 583-589.

[66] Michael G. Jacko, "Physical and Chemical Changes of Organic Disc Pads in Service," *Wear* 46 (1978): 163.

[67] See note 64, *supra*.

Rohl et al. analyzed the wear debris from brake drums of automobiles and found that in general only 3 to 6% by weight of the debris was recognizable asbestos, implying that 94 to 97% of the debris was some other material.[68] They further determined that such asbestos as was present in the wear debris consisted predominantly of short fibres. About 80% of the asbestos fibre in the debris was shorter than 3,750 Angstroms (0.375 microns) in length[69] so that a very small fraction, perhaps 1%, of these fibres would have been longer than 5 microns. Thus, researchers seem to be in agreement that brake wear produces debris that is 6% or less asbestos, and that less than 1% of these asbestos fibres are longer than 5 microns.

Fibre levels experienced during removal of brake wear debris and reported by various authors are summarized in Table 6.12. Most of the readings were derived from optical measurements which means that non-asbestos fibres as well as asbestos fibres were counted. Although the peak levels reported in a number of studies were high (87 f/cc), these levels were realized only intermittently and for brief durations. In contrast, the samples taken over a longer period that included these peak exposures, yielded time-weighted average fibre counts within the range of values found in other occupational exposures to asbestos in Ontario. Furthermore, since we concluded that asbestos constitutes a small fraction of the brake wear debris, it is likely that many of the reported airborne fibres were not asbestos fibres, and that the actual concentrations of asbestos fibres longer than 5 microns experienced during removal of wear debris were below the current 1 f/cc control limit, on a time-weighted average basis.

Table 6.12 also shows airborne fibre levels experienced during modification of new brake linings. Grinding and bevelling new brake linings to allow them to fit the brake drum do not subject the lining to the same high temperatures that are experienced during braking, so physical and chemical alteration of asbestos is unlikely to occur. Thus, the exposure of mechanics during grinding of brake linings resembles more closely the exposure experience of employees engaged in friction products manufacture than it does the exposure of mechanics involved in removing brake wear debris from drums, in that most fibres observed during grinding will be asbestos fibres.

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[68] Arthur N. Rohl et al., "Asbestos Exposure During Brake Lining Maintenance and Repair," *Environmental Research* 12 (1976): 113.

[69] Ibid., Table 2, p. 117.

Table 6.12 shows that bevelling and grinding may cause considerable fibre release. For instance, Lorimer et al. reported dust levels gathered during personal samplings of short duration, analyzed by optical microscope, which ranged from 1.7 to 7.0 f/cc for grinding truck brake linings and 23.7 to 72.0 f/cc for bevelling new linings.[70] The Ontario Ministry of Labour found fibre levels ranging from less than 0.1 f/cc to 15.6 f/cc during machining of brake linings at five brake relining shops in Ontario.[71]

Considering the fibre levels reported in these activities, we believe that the mechanics who regularly grind and bevel brake linings without undertaking proper precautions may be exposed to airborne asbestos fibre concentrations that exceed either the current Ontario chrysotile short-term exposure limit or the TWA control limit, or both.

Medical studies investigating the relationship between brake repair and the incidence of pulmonary disease have consistently demonstrated a greater prevalence of abnormalities among brake repair workers than would be expected in the general population.[72] As well, Newhouse and Thompson, Greenberg and Davies, and A.D. McDonald et al. have all documented cases of mesothelioma in brake repair and automotive mechanics.[73] While it has been suggested that the pattern of disease manifested in brake repair workers parallels that of workers employed in industries with occupational exposure to asbestos, no epidemiological investigation of mortality has been conducted which would either support or refute this proposition.

There are three distinct types of brake repair facilities in Ontario. The first type encompasses the vast majority of the estimated 11,000 establishments in Ontario doing automotive work. Most of these shops are general service stations, independent garages, and franchised new car dealers that will rarely perform more than 8 to 10 brake jobs per week. None of the

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[70] William V. Lorimer et al., "Asbestos Exposure of Brake Repair Workers in the United States," *The Mount Sinai Journal of Medicine* 43:3 (May–June 1976): 207–218.

[71] Letter from Mr. Arthur L. Gladstone, Senior Policy Advisor, Occupational Health and Safety Division, Ontario Ministry of Labour to the Royal Commission on Asbestos, 12 August 1981.

[72] See, for example, M.A. Boillat and M. Lob, "Risk of Asbestosis in Workers Employed in Replacing Automobile Brake Linings," *Schweizerische Medizinische Wochenschrift* 103:39 (1973): 1354–1359; Lorimer et al., "Asbestos Exposure of Brake Repair Workers in the United States," and William J. Nicholson, "Investigation of Health Hazards in Brake Lining Repair and Maintenance Workers Occupationally Exposed to Asbestos," final report to the U.S. National Institute for Occupational Safety and Health, April 1983. (Mimeographed.)

[73] Muriel L. Newhouse and Hilda Thompson, "Mesothelioma of Pleura and Peritoneum Following Exposure to Asbestos in the London Area," *British Journal of Industrial Medicine* 22 (1965): 261–269; Morris Greenberg and T.A. Lloyd Davies, "Mesothelioma Register 1967–68," *British Journal of Industrial Medicine* 31 (1974): 91–104; Alison D. McDonald et al., "Epidemiology of Primary Malignant Mesothelial Tumors in Canada," *Cancer* 26:4 (1970): 914–919.

Table 6.12

Asbestos Exposure During Brake Maintenance

Reference	Operation	Sampling Method/Location (Number)	Sample Period (Minutes)	Fibre Concentration (f/cc)	Peak/ TWA*	Comments
Hickish and Knight[a]	Blowing out dust	Area by side of car (2)	90	1.25	TWA	Four brake blowouts during time
		Area on dust cloud during blowing (2)	90	2.55	TWA	
	Brake cleaning on trucks	Personal (2 men)	450	0.68	TWA	– eight hour shift
			100	7.09	TWA	– brake work
			300	0.08	TWA	– not working on brakes
	Background	Area (AM/PM)				
	– adjacent bay		180	0.28/0.07	TWA	Brakes blown in AM but not in PM
	– 2 bays away		180	0.17/0.07	TWA	
	– garage centre		180	0.49/0.11	TWA	
Knight and Hickish[b]	Blowing dust	Personal	60	5.35		
			60	0.84		
		Personal (2)	2 secs.	87.0	Peak	During blowing
		Area samples adjacent bay	60	0.52		
			60	0.16		
Hatch[c]	Blowing dust	Area samples in dust cloud (7)	few secs.	43 ± 2.1	Peak	Hand-held pump
Lorimer et al.[d]	Blowing dust	Personal 3-5 ft. from operation (4)	2-10	6.6-29.4	Peak	
		Personal 5-10 ft. from operation (3)	2-10	2.0- 4.2	Peak	
		Personal 10-20 ft. from operation (2)	2-10	0.4- 4.8	Peak	
	Blowing dust	Background 10-75 ft. from operation (6)	few secs. -10 mins.	0.1- 0.8	Peak	
	Grinding used truck brakes	Personal (10)	2-10	1.7- 7.0		
		Background 10-60 ft. (5)		0.2- 1.7		
	Bevelling new linings	Personal (5)		23.7-72.0		
		Background 8-30 ft. (4)		0.3- 0.6		

Table 6.12 (continued)

Asbestos Exposure During Brake Maintenance

Reference	Operation	Sampling Method/Location (Number)	Sample Period (Minutes)	Fibre Concentration (f/cc)	Peak/ TWA*	Comments
Rohl et al.[e]	Cleaning drums with dry brush	1-3 ft. from operation (2)	3-8	1.3- 3.6	Peak	
	Background samples 3 minutes after cleaning drums with dry brush	12 ft. (3)		0- 0.2		

Note: *TWA means time-weighted average.

SOURCE: [a] D.E. Hickish and K.L. Knight, "Exposure to Asbestos During Brake Maintenance," *Annals of Occupational Hygiene* 13:1 (January 1970): 17-21.

[b] K.L. Knight and D.E. Hickish, "Investigations into Alternative Forms of Control for Dust Generated During the Cleaning of Brake Assemblies and Drums," *Annals of Occupational Hygiene* 13:1 (January 1970): 37-39.

[c] D. Hatch, "Possible Alternatives to Asbestos as a Friction Material," *Annals of Occupational Hygiene* 13:1 (January 1970): 25-29.

[d] William V. Lorimer et al., "Asbestos Exposure of Brake Repair Workers in the United States," *The Mount Sinai Journal of Medicine* 43:3 (May-June 1976): 207-218.

[e] Arthur N. Rohl et al., "Asbestos Exposure During Brake Lining Maintenance and Repair," *Environmental Research* 12 (1976):110-128.

employees here are organized. The second type of shop is the automotive shops, frequently franchised, specializing exclusively in brake repair work, or brake repair and one or two other specialities. The majority of employees in this type of shop are also not organized. Shops of this type complete on average in excess of 50 brake jobs per week. The exposure of mechanics in these shops is greater than in the shops of the first type simply because of the higher frequency of operations. The third type of shop is the service department for the large automotive fleets. This includes the shops of major trucking companies and urban transit operators. In a large shop of this type, there may be a separate brake department where the workers deal only with brakes. It is in such shops that it is likely that a substantial volume of bevelling and grinding of new brake linings will be performed.

Brake jobs involving the installation of new brake linings in the first two types of shops will seldom require manual modification of the linings. This is because for virtually all passenger vehicles and small trucks undergoing brake repair in these shops, pre-machined linings (i.e., linings which are manufactured to conform to the exact dimensions of the brake drum) are available and only infrequently will manual modification be required to fit the relined shoe to the drum. For those operations using pre-machined linings, it would be unlikely that more than 5% of all relined brake shoes would require manual modifications to fit the drum. Thus, the major opportunity for exposure to brake dust is removed. However, there is still potential for exposure to brake wear debris in these shops.

While pre-machined brake linings are available for the larger vehicles repaired in the third type of shop, some fleet owners stock only one oversized brake lining size and alter the linings to meet the specific size requirements of the brake drums being repaired. In these shops, we would expect that a number of employees would engage continuously in grinding and bevelling of drum brake linings.

Mechanics repairing drum brakes may be protected from dust exposure in two ways. A high efficiency particulate air (HEPA) filtered vacuum may be used to remove wear debris from brakes, instead of blowing the debris out with an air hose. Alternatively, wet cleaning methods may be used either by wiping the drum and assembly with a damp cloth or by directing pressurized water or brake shoe spray into the drum. Bragg discussed the use of an inexpensive spray with a bowl to collect the contaminated liquid. Brake washer systems, including the liquid spray hose and trolley, are available for about $200.[74]

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[74] Bragg, *The Technical Feasibility and Cost of Controlling Workplace Exposure to Asbestos Fibres*, p. 90.

G.2 Shipyard and Railroad Exposure

Shipbuilding and ship repair constitute another activity in which workers may be exposed to airborne asbestos fibres. Since before World War II, fireproofing and thermal insulation, similar to the insulation sprayed in buildings in the 1950s and later years, was sprayed onto metal surfaces and otherwise installed in ships. Exposed workers would include those applying the insulation, those working on the ship in the vicinity of the spraying, and some trades who worked on the ships after the insulation was installed. We do not have data on the intensity of shipyard worker exposures, but they might be comparable to those of insulation workers, discussed in Chapters 9 and 10. Table 6.13 shows employment in shipyard work in Ontario from 1939 to the present. This employment grew substantially during World War II, reaching a peak of almost 9,000 workers in 1943–1944. We have no way of knowing what proportion of this workforce was exposed to asbestos. By the 1950s, employment was about one-quarter of its peak level, and it is now less than 2,000 workers. War-time work included the construction of corvettes and minesweepers for the Canadian, British, and American Navies.

Railroad vehicle construction and maintenance work has also been a source of worker exposure to asbestos. The boilers and fireboxes on steam locomotives were frequently covered with asbestos block insulation, similar to the insulation used on steam boilers in buildings. (See Chapters 9 and 10.) Construction of steam locomotives therefore requires the installation of asbestos insulating block. From time to time, the locomotive boiler would have to be re-insulated, which involved removing the metal jacketing around the insulation, removing the asbestos block insulation, repairing the boiler and piping as necessary, and then re-insulating the boiler and piping. All workers involved in these activities suffered some possible exposure to asbestos. We have not found data on the intensity of this exposure, but it might be of the same order of magnitude as that for insulation workers dealing with boilers and pipes in buildings, which can be quite considerable. (See Chapters 9 and 10.) In the United States, asbestos-related disease has been found in railroad workers engaged in locomotive maintenance.[75] We have not found estimates of the number of railway workers so exposed in Ontario.

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[75] Martin-Jose Sepulveda, "Roentgenographic Evidence of Asbestos Exposure in a Select Population of Railroad Workers," Morgantown, West Virginia, NIOSH, July 1982. (Mimeographed.); and Ronald L. Motley and Richard H. Middleton, Jr. "Asbestos Disease Among Railroad Workers: Legacy of the Laggin' Wagon," *Trial* (December 1981): 39–44.

Table 6.13

Employment in Ontario Shipyards, 1938-1980 (Production Workers)

Year	Number Employed
1938	551
1939	430
1940	NA*
1941	NA
1942	6,283
1943	8,914
1944	8,905
1945	5,103
1946	2,246
1947	2,171
1948	2,872
1949	2,308
1950-1954 (Average)	2,944
1955-1959 (Average)	1,819
1960-1964 (Average)	1,857
1965-1967 (Average)	2,060
1968-1973	NA
1974	1,668
1975-1979 (Average)	1,814
1980	1,942

Note: *NA means data not available.

SOURCE: Statistics Canada, Catalogue 42-206, *Employees and Their Earnings in the Shipbuilding Industry.*
For 1937-1948, data are from Statistics Canada, Census of Manufactures.

Chapter 7 Control Limits for Fixed Place Industry

A. Introduction

In this chapter we will develop our recommendations concerning the regulation of asbestos in fixed workplaces by way of prohibition or of control limits governing worker exposure. The first step is to analyze, in Section B, the measurement methods used to determine worker exposure to asbestos. In Section C we use some of the epidemiological studies discussed in Chapter 5, and models developed in the Appendix to the present chapter, to estimate the risks of respiratory cancer that might result from applying alternative control limits. In Section D we review current control limits in several jurisdictions. In Section E we consider the criteria that should be used to determine workplace control limits in general, and asbestos control limits in particular. In Section F we apply these criteria to our quantitative risk assessment, enriching this assessment with other data and conclusions from Chapter 5, and we prescribe regulatory responses that differ for different industrial processes. Finally, in Section G we discuss the special case of regulating the asbestos exposures of automotive brake mechanics.

B. Measurement of Asbestos Fibres in the Workplace

B.1 Objectives of Workplace Measurement

The purposes of measuring workplace asbestos dust levels are to determine the exposure of workers to health-related risks and to determine compliance with health-related exposure control limits. Thus, the design of a measurement technique should depend upon the answers to some of the medical questions considered in Chapter 5. It is generally accepted that the health risk from asbestos fibre exposure increases with the quantity of asbestos to which the individual is exposed, although there is considerable debate on whether the relationship is linear or otherwise. We have discussed whether the degree of risk presented by a given fibre concentration in the air is affected by the type of fibre or the length of fibre. Because we have accepted both of these propositions, it would be useful to measure both length and fibre type. There is debate on whether health risks depend only upon the total cumulative exposure of the individual, or whether they depend upon the time profile of that exposure. In particular, some researchers believe that a given dose in a short, intense concentration might be more harmful than the same dose spread over a longer period of time. Thus, it may be useful to gather information on the time profile of worker exposure.

While a measurement technique might usefully gather all of the above information, it should be sufficiently economical to allow reasonably frequent measurement, and it should provide reasonable accuracy (ability to provide the correct measurement) and precision (ability to repeat a given measurement when there is no change in the underlying phenomenon). The measurement technique should also allow one to compare current measurements with previous measurements to determine whether exposures are rising or falling. Finally, a technique that allows the underlying information to be preserved for a long period of time will facilitate future studies of the data.

We have heard many arguments about the limitations of current techniques for measuring asbestos fibre concentrations at low concentration levels, an issue that will be discussed in detail below. We believe that if health and other considerations require a control limit that is below the level that can be adequately measured by current technology, the solution is not to raise the control limit to a level that can be measured, but rather to consider prohibition of any exposure to the substance until such time as measurement technology is available that can be used to enforce compliance with the desired control limit. This should encourage the development of such improved measurement technology.

B.2 The Membrane Filter Method

Historically, occupational exposure to asbestos was based upon visual counts of asbestos dust particles using various dust sampling techniques. In the late 1960s, the technology of measurement improved and changed from a count of dust particles to an actual count of asbestos fibres. For measurement purposes, an asbestos fibre was defined in accordance with common international practice as a fibre longer than 5 microns,[1] whose length to diameter ratio (aspect ratio) was at least 3 to 1. This definition originated in the United Kingdom, having been somewhat arbitrarily selected by three asbestos manufacturers who collaborated on the matter. It was apparently chosen to facilitate counting using the optical microscope, and because it was thought that the development of asbestosis was related to longer fibres. Because some researchers conclude that fibres with diameters exceeding about 3.5 microns are not respirable, some versions of the measurement rules specify a maximum diameter of 3 microns or 5 microns.

In the membrane filter method, a volume of air is drawn through a membrane filter by a pump. If the air is drawn from near the worker's face, it is a personal sample, otherwise it is an area sample. Asbestos fibres and other solids are collected on the membrane filter. The filter is removed from the sampler and taken to a laboratory where it is made transparent (cleared), mounted on a slide, and inserted into a microscope, usually a phase contrast microscope (PCM). An operator examines portions (fields) of the filter under the microscope and counts the number of fibres present, using elaborate rules to determine whether an object should be counted as a fibre. The average fibre concentration over the entire filter can be estimated from the number of fibres in the fields actually counted, and from this the concentration of fibres in the air where the sample is taken can be estimated.

The membrane filter method has a number of limitations as a means of determining the exposure of workers to asbestos fibres. However, no alternative measurement method seems to be sufficiently attractive, according to the criteria suggested above, to be a logical candidate for replacing the membrane filter method. Electron microscope methods may provide greater accuracy or better fibre identification, but they are currently considerably more expensive. In addition, because the electron microscope can detect fibres not visible in the optical microscope, electron microscope counts are not directly comparable to the optical fibre count data accumulated over the last fifteen years and to control limits enforced by optical fibre counts. However, it is possible to compare fibre counts using both methods if appropriate adjustments are made. (See Chapter 9, Section C.) Another method for measuring low concentrations of asbestos fibres in the air is gravimetric or mass measurement, discussed in Chapter 9, Section C. While this mass measurement might be applied to workplaces with low fibre concentrations, there is again the problem of converting mass measurements to an equivalent optical fibre count. Without such a conversion it is impossible to know whether a gravimetric reading represents a greater or lesser exposure than the current control limit expressed in fibres per cubic centimetre, measured optically. Furthermore, there is no generally accepted method for analyzing gravimetric samples, and each researcher currently uses a different method, so that their results are not comparable. Thus, it seems appropriate that the regulation of asbestos in the workplace continue for the near future to rely on the membrane filter method.[2]

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[1] Eric J. Chatfield, *Measurement of Asbestos Fibre Concentrations in Workplace Atmospheres*, Royal Commission on Asbestos Study Series, no. 9 (Toronto: Royal Commission on Asbestos, 1982), p. 4. The correct technical term for one-millionth of a metre is micrometre, but we will use the more familiar term, micron.

In Section F of this chapter we conclude that in some cases the control limit for asbestos should be set at a concentration below that which can reliably be measured using the membrane filter method. This causes such uses of asbestos to be prohibited until measurement technology is available that allows these control limits to be reliably enforced. If the industry believes that the low control limits can be achieved, and that there is substantial economic value to using asbestos in this way, then industry may develop improved measurement techniques that would demonstrate compliance with these control limits. Section 17 of the Regulation Respecting Asbestos states that ". . . the methods and procedures that may be used or adopted may vary from the codes issued by the Ministry if the protection afforded thereby or the factors of accuracy and precision used or adopted are equal to or exceed the protection or the factors of accuracy and precision in the codes issued by the Ministry."[3] We interpret this section to mean that the Ministry would allow some fibre measurement method other than the membrane filter method specified in the Code for Measuring Airborne Asbestos Fibres to be used if the Ministry was satisfied that the alternative measurement technique was appropriate.[4] We therefore recommend that:

7.1 ***Whenever control limits that cannot be enforced by present measurement techniques are adopted, the Ministry of Labour should emphasize that section 17 of the Regulation Respecting Asbestos allows the substitution of alternative measurement techniques if the Ministry is convinced that the substitute techniques satisfy the criteria of section 17 of the Regulation.***

B.3 Recommended Improvements to the Membrane Filter Method

In a study prepared for this Commission, Dr. Eric J. Chatfield discussed some of the problems with the existing application of the membrane filter method in Ontario.[5] The Ontario Ministry of Labour method of preparing the membrane filter to produce microscope slides uses dimethyl phthalate and dimethyl oxalate and is required by the Code for Measuring Airborne Asbestos Fibres, section 2(2)(f). This is the mounting method used by the National Institute for Occupational Safety and Health (NIOSH) and the Occupational Safety and Health Administration (OSHA) in the United States. Unfortunately, the method does not yield a durable slide, and the fibres must be counted within two days, or they cannot be counted at all. In contrast, the Asbestos International Association (AIA) preparation method, using acetone-Triacetin, produces a durable slide that also yields better fibre resolution, allowing more of the fibres to be seen and counted.[6] A minor drawback to the acetone-Triacetin method is that it requires the use of a fume hood and must therefore be performed in a laboratory.[7] We believe that durability and resolution are important. We therefore recommend that:

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[2] Ibid., sec. 5.1.

[3] Regulation Respecting Asbestos, O. Reg. 570/82, made under the *Occupational Health and Safety Act*, R.S.O. 1980, c. 321.

[4] See Code for Measuring Airborne Asbestos Fibres, in Regulation Respecting Asbestos.

*7.2 **The Ministry of Labour mounting method should be amended to replace the dimethyl phthalate and dimethyl oxalate preparation step with the acetone-Triacetin preparation step.***

The improved resolution of this preparation should improve the accuracy of fibre counting in Ontario. We believe that there would be some value in preserving counted slides for a period of time for use in auditing the accuracy of reported fibre counts, as possible evidence in any prosecutions for violation of the control limit, and for research that requires more information than is provided on the data sheets prepared by fibre counters. For example, old slides could provide a data base for analysis of the fibre dimensions found in different workplaces. However, because under section 15(1) of the Regulation Respecting Asbestos the records of work exposures must be maintained for up to forty years, we doubt that the permanent storage of slides is necessary. Storage for a five-year period should provide sufficient time for preserved slides to serve their purposes. We therefore recommend that:

*7.3 **The Ministry of Labour should require that all slides and remaining filter segments that represent workplace exposures in Ontario, whether counted by the Ministry of Labour or by a private firm, be catalogued and stored in Ontario for a period of five years after the sample is taken.***

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[5] Chatfield, *Measurement of Asbestos Fibre Concentrations in Workplace Atmospheres*, sec. 2.

[6] Ibid., p. 8.

[7] Gyan S. Rajhans and John L. Sullivan, *Asbestos Sampling and Analysis* (Ann Arbor, Michigan: Ann Arbor Science Publishers Inc., 1981), p. 113.

Dr. Chatfield has stated that "It has been demonstrated . . . that incorrect alignment of the phase contrast microscope can result in significant reductions in fibre counts . . . . The AIA method and the proposed ISO/DIS [International Organization for Standardization/Draft International Standard] method both clearly specify the use of test slides to ensure adequate microscope resolution. The other published methods do not."[8] The Regulation Respecting Asbestos does not incorporate any test of microscope resolution. We recommend that:

7.4 The Ministry of Labour method should be modified to incorporate tests of microscope resolution.

Dr. Chatfield indicated that the selection of the microscope graticule can influence the accuracy of the fibre count.[9] Only the Walton-Beckett graticule has been designed specifically for fibre counting. It is the only graticule that does not demand visual interpolation by the operator to determine whether fibres exceed the critical dimensions. In both the AIA and ISO/DIS methods, this graticule has been adopted as the standard. The Code for Measuring Airborne Asbestos Fibres requires the use of the Patterson Globe and Circle reticle.[10] We recommend that:

7.5 The Ministry of Labour should specify the Walton-Beckett graticule exclusively in the Code for Measuring Airborne Asbestos Fibres.

To control for variability in counting among operators from one time to another, the U.S. Public Health Service and the National Institute for Occupational Safety and Health (USPHS/NIOSH) recommend that one of 10 samples should be recounted blind, so that the operator does not know that the slide is being recounted, either by the same operator or by a second operator. This provides a specific test of the repeatability of the fibre counting.

The Ministry of Labour requires recounting of only one sample in 20, or one per batch, whichever is lower, and allows recounting by the same operator.[11] Dr. Chatfield has noted that recounting by the same operator ignores the most significant variability factor.[12] We recommend that:

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[8] Chatfield, *Measurement of Asbestos Fibre Concentrations in Workplace Atmospheres*, p. 9.

[9] Ibid., pp. 59–60.

[10] Code for Measuring Airborne Asbestos Fibres, s. 2(2)(a)(iv).

[11] Ibid., s. 4(4).

[12] Chatfield, *Measurement of Asbestos Fibre Concentrations in Workplace Atmospheres*, p. 10.

7.6 ***The Ministry of Labour method should be modified to provide a more rigid system of quality assurance, including the use of blank filters and recounting by other operators.***

Because there are many objects of a variety of shapes on a filter, it is important to have a consistent set of rules telling operators what should be counted as a fibre and what should not. While there is room for much debate as to what should properly be counted, there is some advantage in following internationally accepted procedures on this matter. According to Rajhans and Sullivan, the AIA reference method has incorporated the best components of counting methods from the United Kingdom, the United States, and Australia, providing detailed fibre counting criteria that should reduce counting variability.[13] We recommend that:

7.7 ***The fibre counting criteria published in the Asbestos International Association's (AIA) reference method should be adopted by the Ontario Ministry of Labour as an interim measure since they represent a current level of international consensus. If and when agreement is reached on counting criteria to be adopted for the International Organization for Standardization/Draft International Standard (ISO/DIS) method, these should then be considered for adoption by Ontario.***

Because of subjective elements in determining what to count as a fibre, and because of variations in analytical and laboratory techniques, there may be considerable differences in the number of fibres counted on a given slide by various operators or various laboratories. These variations can occur even when the operators are nominally using the same counting rules and techniques. Dr. Chatfield reported that when a set of filters is distributed to different laboratories, or even to different operators within a given laboratory, the resulting fibre counts are highly variable.[14] Variations of a factor of 10 have occurred between individual counters. Systematic variations of a factor of 3 have been observed between laboratories that were apparently using identical analytical procedures. Dr. Chatfield reported on an international round-robin trial in which maximum fibre counts ranged from 2.8 to 6.2 times as large as the lowest fibre count for the same filter.[15] All of these measurement problems become more serious as the workplace control limit is reduced and the fibre levels that must be measured decline.

The only means of reducing these variations is to conduct round-robin trials in which a set of slides is sent around to various counters, the fibres counted, and the results reported. Where substantial discrepancies exist among individuals or laboratories, some discussion must take place in which the source of the discrepancy is identified, and agreement is reached as to how to eliminate it. Dr. Chatfield has suggested that the use of a central reference system as adopted in the United Kingdom and in Sweden can reduce the inter-laboratory variability.[16] The Ministry of Labour could select its own laboratory, some other Ontario laboratory, or conceivably a non-Ontario laboratory to act as a central reference laboratory. The central reference system would conduct round-robin trials among fibre counters and certify those whose performance is acceptably close to that of the average of the group or to that of some reference operator or operators.

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[13] Rajhans and Sullivan, *Asbestos Sampling and Analysis*, p. 101.

[14] Chatfield, *Measurement of Asbestos Fibre Concentrations in Workplace Atmospheres*, pp. 24–25.

[15] Ibid., Table 3, p. 25.

Every attempt should be made to include worker representatives from Ontario asbestos manufacturing plants among such certified operators where the volume of measurements warrants it. We believe that this will help to ensure that the workers whose health depends upon the maintenance of low asbestos fibre levels will view the measurements taken in their workplaces as reliable. We therefore recommend that:

7.8 The Ministry of Labour should identify a laboratory to take responsibility for standardizing methods of asbestos fibre counting for workplace samples in Ontario and to certify individuals whose performance is judged by this laboratory to be satisfactory. Every attempt should be made to include worker representatives from Ontario asbestos manufacturing plants among such certified operators where the volume of measurement warrants it.

B.4 Sampling and Measurement Errors and Compliance

The purpose of workplace measurements is to determine the true total exposure over time of workers to asbestos fibres in the air.[17] The actual measurement is based upon samples from a few workers over a few hours out of their working year. Thus, the measurements that are taken provide only an estimate of the true exposure of the worker. Sources of error between the measurement and the worker's actual exposure include the following:

(i) The period sampled may not represent the exposure of the worker himself.

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[16] Ibid., p. 34.

[17] Ideally, workplace measurements should determine the true exposure over time of workers to those asbestos fibres that are most toxic. Any exposure measure is only an index of the hazard actually experienced.

(ii) The sampling method may not contain a representative sample of the air the worker breathes.

(iii) The sampling pump may not be properly calibrated or operated.

(iv) The preparation of the filter may lose some fibres.

(v) Because only portions of a given filter are actually counted, the fields counted may not accurately represent the fibre density on the entire filter.

(vi) The operator's fibre count may over- or underestimate the actual number of fibres present.

Thus, a single fibre count is only an estimate, and perhaps not a very good estimate, of the actual exposure of the worker to asbestos fibres.

One solution to this problem is to obtain more samples. If each worker were monitored continuously, then the first problem would be solved and there would be a sufficient number of measurements so that the other errors might tend to cancel each other out. This is impractical, however, because of the cost and inconvenience to the worker, and thus we must assume that the total number of measurements taken will continue to be rather limited.

(a) The Confidence Interval of a Measurement

This leaves the question of what one can properly infer from the analysis of one or more samples at a work station. The fibre count from a single sample is the best estimate of the actual exposure of the worker. The average fibre count from a number of samples is the best estimate of the average exposure at the work station.[18] The accuracy of this estimate can be described by the "95% confidence limit" of the estimate. The 95% confidence limit is the range of values within which the actual exposure will be contained 95% of the time, or 19 out of 20 times. If the measurement is very precise, then the 95% confidence limit may be a very narrow range of values around the sample mean. If the measurement is highly imprecise, then the 95% confidence interval may be a very wide range around that number. A study by the Air Monitoring Committee of the Asbestos Information Association/North America (AIA/NA) has shown that if a single 8-hour sample yields a fibre count of 1 fibre per cubic centimetre (f/cc), then it may be 95% certain that the true fibre count is between 0.26 and 2.73 f/cc.[19] (See Figure 7.1.)

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[18] The best estimate of the average exposure U, given a set of sample readings x, is obtained first by taking the log of each sample reading, since the exposures are approximately log-normally distributed. Let the log of x be called y. The mean of the sample logs is Y, and the variance of the y is s^2. The best estimate of the true average exposure U is: $U = \exp. (Y + {}^1/_2\, s^2)$. See J.A. Greenwood and H.O. Hartley, *Guide to Tables in Mathematical Statistics* (Princeton, N.J.: Princeton University Press, 1962), p. 412. See also, John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," *Annals of Occupational Hygiene* 26:1–4 (1982): 872.

We have seen that a single fibre measurement may give a very imprecise estimate of the true exposure of the worker. The estimate of the worker's actual exposure can be improved by taking additional samples. The width of the confidence limit for the estimate of the true exposure is inversely proportional to the square root of the number of measurements taken, so that quadrupling the number of measurements will divide the confidence limit approximately in half. (See the dotted line in Figure 7.1.)[20] If some of the variability in the fibre measurements arises specifically from errors in fibre counting, then repeated counting of a given filter may allow some improvement in the confidence interval of the estimate. If one is interested in estimating the worker's average exposure, adding independent sample measurements or fibre counts is an important means of improving the precision of this estimate.

(b) The Detection Limit

The detection limit of a measurement method is the lowest concentration at which the method may reliably be used to determine whether the contaminant is present or not. The errors discussed above contribute to causing the detection limit to be some positive concentration and not zero. The possibility that asbestos fibres may be found on slides even when there was no asbestos in the air means that low fibre counts may erroneously be interpreted as demonstrating that asbestos was in the air. Even blank slides will often yield a fibre count of a few fibres in 100 fields. The AIA/NA study concluded that the detection limit for the membrane filter method is no lower than 0.1 f/cc.[21]

The detection limit is the lowest level for which the *presence* of asbestos can reliably be measured. The *quantity* of asbestos present can only be estimated accurately at concentrations well above the detection limit. Thus, the lower limit for accurate measurement of asbestos fibre concentrations is well above 0.1 f/cc. Since for any true exposure level, some measured fibre

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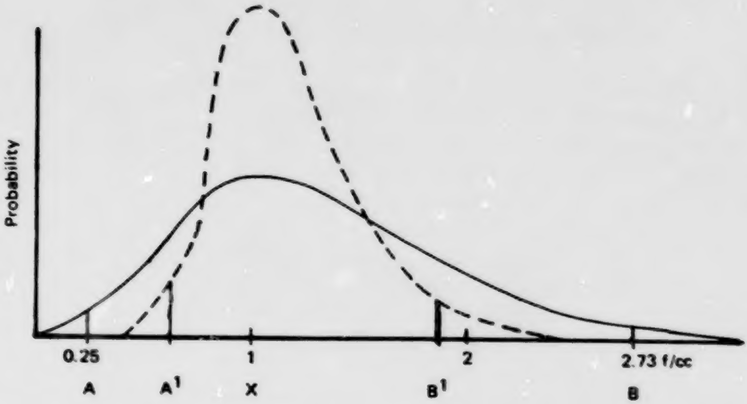
[19] Asbestos Information Association/North America, *A Study of the Empirical Precision of Airborne Asbestos Concentration Measurements in the Workplace by the Membrane Filter Method*, prepared by the Air Monitoring Committee of the AIA/NA, Dr. Harrison B. Rhodes, Chairman (Arlington, VA: AIA/NA, June 1983), Table 8.1.

[20] The confidence limit would be divided precisely in half if the measurements were independent and their probability distribution were normal.

[21] Asbestos Information Association/North America, *A Study of the Empirical Precision of Airborne Asbestos Concentration Measurements in the Workplace by the Membrane Filter Method*, pp. 8-22.

Figure 7.1

Distribution of Probable Values of True Exposure Given a Measurement X



Notes: A, A^1 = lower confidence limit.
B, B^1 = upper confidence limit.

counts will be above and some below the true level, a firm that wishes to be in compliance with a control limit 95% of the time must produce an average fibre level that is below the control limit. Thus, the lowest control limit for which compliance can reliably be proved must be well above the detection limit.

(c) Average Exposures Under Control Limits

What is the relationship between the average exposure of a worker and a control limit that is enforced? Dr. Chatfield has shown that given the variability of fibre readings taken in an Ontario brake plant, if the plant were to exceed the control limit no more than 5% of the time, its average fibre concentration would have to be between one-half and one-quarter of the control limit or less, depending on the variability of these measurements.[22] (See Figure 7.2.) The AIA / NA study demonstrated that given the variability of its data, in order to ensure that the upper confidence limit was below 1 f/cc, that is, to ensure that there was less than a 2.5% chance that the true fibre level exceeds 1 f/cc, a single 8-hour time-weighted average (TWA) sample would have to be about 0.30 f/cc or less.[23] Table 6.7 in Chapter 6 shows that average fibre counts could lie between 0.5 and 1.0 f/cc when 10 to 20% of those counts exceeded 2 f/cc. The United Kingdom Advisory Committee on Asbestos reported that in friction products a *median* fibre count of 0.2 f/cc could yield 95% of all counts less than 2 f/cc, with 85% less than 1 f/cc.[24] Thus, if enforcement of a control limit of 1 f/cc meant that the Ministry of Labour allowed 10 to 20% of all observed fibre counts to exceed 1 f/cc, the average worker's exposure might be between 0.25 and 0.5 f/cc. With more stringent enforcement, so that 5% or less of all fibre counts exceeded the 1 f/cc control limit, the average worker exposure might be less than 0.25 f/cc.

Dr. Chatfield concluded that for a control limit of 0.2 f/cc, compliance cannot be demonstrated on the basis of a 90-minute air sample.[25] Compliance with the 1 f/cc control limit can be demonstrated if sufficient quality control is used in the analytical techniques. Dr. Chatfield regards the demonstration of compliance at the 0.5 f/cc level as possible but somewhat uncertain.[26] Dr. Gerald R. Chase and Dr. Harrison B. Rhodes

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[22] Chatfield, *Measurement of Asbestos Fibre Concentrations in Workplace Atmospheres,* Table 7, p. 43.

[23] Asbestos Information Association / North America, *A Study of the Empirical Precision of Airborne Asbestos Concentration Measurements in the Workplace by the Membrane Filter Method,* Table 8.1.

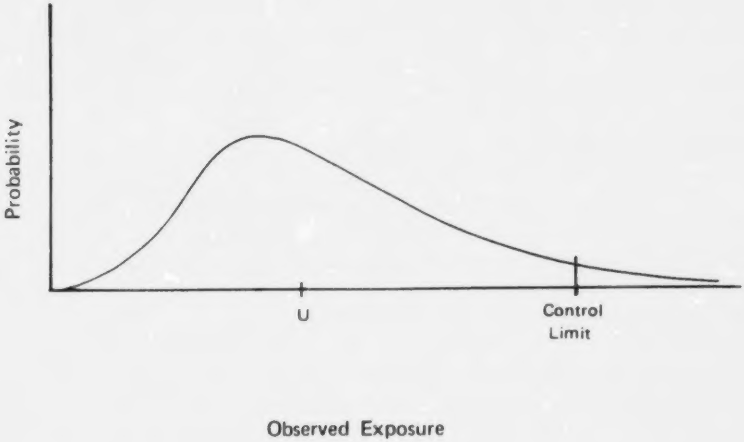
[24] U.K., Advisory Committee on Asbestos, *Asbestos — Volume I: Final Report of the Advisory Committee* (Simpson Report), William J. Simpson, Chairman (London: Her Majesty's Stationery Office, 1979), Figure 1, p. 17.

[25] Chatfield, *Measurement of Asbestos Fibre Concentrations in Workplace Atmospheres,* p. 44.

[26] Ibid.

Figure 7.2

Probability of Observing Various Exposures Given Average Exposure U



agreed that the lowest control limit that would allow an employer to be 95% certain that no more than 5% of employee exposures exceeded the control limit would be between 0.5 and 1.0 f/cc.[27]

We, therefore, conclude that using the membrane filter method, it is not possible to use single samples to demonstrate reliably compliance with a control limit of 0.1 f/cc and 0.2 f/cc. We conclude that demonstrating compliance with a control limit of 0.5 f/cc might be possible with present analytical methods, but is subject to considerable uncertainty. Averaging a number of samples might be required to test compliance reliably at the 0.5 fibre level. Demonstrating compliance with a control limit of 1.0 f/cc is possible using single samples and present measurement methods.

(d) Sampling Frequency for Enforcing Control Limits

The Ministry of Labour has recognized the variability in the exposure of individual workers over the course of a typical work week. The Code for Measuring Airborne Asbestos Fibres in the Regulation Respecting Asbestos states that a sufficient number of samples of a worker should be taken to calculate a time-weighted average concentration, and these samples must be representative of the worker's exposure.[28] The time-weighted average, to which the control limits of section 4 of the Regulation Respecting Asbestos are applied, are to be calculated as a weighted average of these sample exposures, where each exposure is weighted by the percentage of the week that the worker spends in activity.[29]

Most jurisdictions use a single measurement of the worker's exposure, typically based on an air sample gathered over a period of 1 to 4 hours, to determine whether the control limit has been exceeded or not. We see both advantages and disadvantages to the Ministry of Labour's calculation of a time-weighted average based upon one or more independent samples. In theory, the use of multiple samples allows an accurate estimate of the worker's exposure over the course of a week, since exposure readings may be taken during each of several activities the worker may engage in, and each such exposure can be given a weighting according to how important that activity is over the course of the work week. Thus, if a worker performs a dirty job for 30 hours and a clean job for 10 hours, a 2-hour reading taken during each type of job yields a TWA that reflects the predominance of time spent in the dirty activity. Furthermore, since multiple samples are used to calculate the TWA, the variability in the TWA as an estimate of the worker's true exposure is reduced.

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[27] Ontario, Royal Commission on Asbestos, Exhibit II-39, Tab 11 [hereafter RCA Exhibit], in Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Evidence of Dr. Gerald R. Chase and Dr. Harrison B. Rhodes, 14 August 1981, Volume no. 27, p. 22: Gerald R. Chase and Harrison B. Rhodes, "Measurement of Asbestos Levels in the Workplace," [Arlington, VA], Asbestos Information Association/North America, 14 August 1981. (Mimeographed.)

[28] Code for Measuring Airborne Asbestos Fibres, s. 3(3)(i).

[29] The Schedule following the Regulation Respecting Asbestos provides in ss. 4 and 5 for the calculation of a time-weighted average:

4. The weekly exposure shall be calculated as follows:
$C_1T_1 + C_2T_2 + \ldots + C_nT_n$ = cumulative weekly exposure, where C_1 is the concentration found in an air sample and T_1 is the total time in hours to which the worker is taken to be exposed to concentration C_1 in a week.
5. The time-weighted average exposure shall be calculated by dividing the cumulative weekly exposure by 40.

On the other hand, the time weighting which is theoretically attractive raises practical problems. Some concern has been expressed to this Commission that it will be difficult to determine the time weightings, and that industry may choose these in a way that will underestimate the time-weighted average. Prosecutions may be more difficult because of the complexity of the calculation. Worker interpretation of exposure data may be impeded because of the complexity of the calculation. Furthermore, since the variability of the time-weighted average is less than when single measurements are used, the average worker's exposure may be somewhat closer to the control limit than if single exposures were used for enforcement.

On balance, we have not heard sufficient objections to the Ministry's use of multiple samples for calculating a time-weighted average to lead us to reject this procedure as a basis for enforcing the control limit. However, we urge the Ministry to monitor the field experience with this measurement method and to determine whether the problems raised above are in fact serious. If they are, the Ministry should consider reverting to the use of individual samples to enforce the control limit. In its deliberations, the Ministry should find some guidance in the statistical analysis of sampling strategies contained in a manual published by the U.S. National Institute for Occupational Safety and Health.[30] Furthermore, in Section F of this chapter we endorse control limits on the assumption that average worker exposures will be less than or equal to half of the control limit. If the use of multiple samples in calculating the time-weighted average should allow average worker exposures to rise above half of the control limit, then the Ministry should take action to maintain those exposures at half the control limit or below.

Whether the worker's time-weighted average exposure is to be calculated from multiple samples, or from a single sample, there is still the question of how often such a time-weighted average should be determined for each worker. Should each worker be measured once a year, once a month, or once a week? Should some workers or work stations be measured more than others? The Regulation Respecting Asbestos, and the associated codes, give no guidance on this issue. We can see many factors affecting the choice of a measurement frequency. Measurement is more instructive where the exposures are highly variable than where they are steady. Measurement information is more useful where policies are available to reduce exposures than where no fruitful action can be taken as a result of learning actual exposure levels. If one is particularly interested in detecting control limit violations, then measurements should be more frequent where high readings are suspected. If one is interested in calculating average exposures, then measurements should be randomly distributed among workers and time periods. Measurements might be distributed among plant processes in proportion to the number of workers exposed in each process. More measurements will provide better information, but will raise the cost of the monitoring programme. We do not find in these multiple factors a basis for providing further guidance on the question of measurement frequency in the Regulation Respecting Asbestos. Perhaps the joint health and safety committee, in its participation in a monitoring programme, should consider the objectives it believes are important in its plant and should work to ensure that these are reflected in the choice of measurement frequency. More than this is needed, however, if we are to be confident that the monitoring programme is adequate.

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[30] Nelson A. Leidel, Kenneth A. Busch, and Jeremiah R. Lynch, *Occupational Exposure Sampling Strategy Manual* (Cincinnati, Ohio: U.S. Department of Health, Education and Welfare, National Institute for Occupational Safety and Health, 1977), chap. 3.

We are convinced that the design of a sampling strategy is at least in part a statistical problem. The manual published by the U.S. NIOSH analyzed statistically the frequency and type of measurement necessary in order to achieve particular levels of confidence in estimating average or maximum worker exposures.[31] This manual was written as a guide for enforcing U.S. occupational health legislation. The principles presented, however, have some general applicability and may be useful for enforcing the Regulation Respecting Asbestos and other occupational health regulations in Ontario. The manual defined levels of accuracy that may be achieved by occupational monitoring, providing methodologies for determining how many measurements are required in order to achieve that degree of accuracy. We believe as a matter of principle that the monitoring programmes required for determining worker exposures to asbestos, and probably other toxic materials in the workplace, should be designed to fulfill some minimum requirements regarding the accuracy of the average exposures that may be calculated from those measurements and the likelihood that particularly high worker exposures have been detected. Achieving this end would require that the regulations specify, in some statistical fashion, the goals to be achieved by a monitoring programme. This Commission has not heard sufficient evidence on this issue to formulate a recommendation for the precise form that such a regulation should take.

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[31] Ibid.

We are, however, concerned by the enormous latitude provided by the Regulation Respecting Asbestos with regard to sampling, and we believe that this should be made more precise. We therefore recommend that:

7.9 The Ministry of Labour should undertake a study of the relationship between the number of air samples taken and the distribution of those air samples among workers, work stations, and periods of the day or week, in order to determine the minimum requirements for an air sampling programme that will provide reasonable confidence that the data collected adequately reflect the maximum worker exposures in a workplace, the average exposure of those workers, and those work stations where particularly intensive monitoring may be desirable. This study should incorporate such statistical analyses as are necessary for the task. Upon completing the study, the Ministry should amend the Regulation Respecting Asbestos to provide more specific direction regarding the sampling programme that will satisfy that Regulation.

(e) Sources of Bias in Measurements

This discussion has assumed that all measurement errors are random and are equally likely to overestimate or underestimate the true exposure. However, a survey by Professor Sally Luce and Professor Gene Swimmer, undertaken for this Commission, revealed worker allegations that recorded fibre counts underestimated true fibre concentrations.[32] The primary reasons offered were that the time and place of sampling were not representative of actual worker exposure. It was suggested that the dirtiest operations might be performed on the night shift when samples were never taken. It was suggested that the dirtiest machines were switched off during sampling periods. It was suggested that the plant might be cleaned up in anticipation of the visit of an inspector. While we cannot estimate the quantitative significance of these practices, we believe that it is prudent to assume that actual worker exposures may be somewhat understated by the analysis of air samples taken by the Ministry of Labour alone or by management if there is no effective worker input.

While future enforcement practices are uncertain, we conclude from the above analysis that the imposition of any control limit that is enforced so that only 10% or so of individual samples exceed the control limit will permit worker exposures that, over the course of a few weeks or months, will average half the amount of the control limit. Less strict enforcement of a control limit would allow greater worker exposures. If the average exposure were as high as the control limit, then as many as one-half of all individual samples taken would exceed the control limit, and we see no evidence that this has occurred regularly in any Ontario workplace. The data of Chapter 6, Table 6.7 show that since 1972 the average number of samples exceeding the then existing 2 f/cc guideline ranged from 50% down to zero, with most industries reporting between 10 and 25% of samples exceeding the guideline. We believe that it is unlikely that worker exposures will exceed, on average, the control limit itself, unless enforcement is abandoned completely. Thus, when assessing the health effects of a control limit, we will calculate the disease resulting from a normal exposure, assuming an average of one-half the amount allowed by the control limit, and from a high exposure, assuming that the exposure equals the control limit itself.

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[32] Sally Luce and Gene Swimmer, *Worker Attitudes About Health and Safety in Three Asbestos Brake Manufacturing Plants*, Royal Commission on Asbestos Study Series, no. 6 (Toronto: Royal Commission on Asbestos, 1982).

B.5 Measuring Fibre Size

The size of respirable airborne asbestos fibres in the workplace may range up to 3 to 5 microns in diameter and up to 50 or perhaps 100 microns in length. This range of sizes immediately presents problems for measuring fibre concentration: what size fibres should be counted? We measure asbestos fibre concentrations in order to predict possible health effects. Thus, ideally, we would like to measure those fibres that are particularly relevant for health effects, and to ignore all other fibres. The practical problems of pursuing this objective can be seen by considering two examples.

First, suppose that all asbestos fibres have the same effect regardless of size. In this case, counting the concentration of all asbestos fibres in the air would give us a measure that was proportional to the health effect of those fibres. It is impossible to count all fibres, however, because some are too small to be seen. The PCM cannot detect fibres thinner than 0.2 to 0.4 microns in diameter.[33] The transmission electron microscope (TEM), on the other hand, can detect unit fibrils of chrysotile, meaning that virtually all chrysotile fibres can be detected by TEM, if one is sufficiently patient to search for the small ones. The rules for counting fibres using a PCM exclude fibres shorter than 5 microns, or with an aspect ratio less than 3 to 1. Figure 7.3 shows a typical distribution of fibre sizes that might be present in the atmosphere and the much lesser distribution of fibre sizes that could be detected using optical methods. The shaded area under the optically visible line, above 5 microns in length, indicates the fibres that would actually be counted under current counting rules. This means in practice we count only a fraction of all airborne fibres, when using PCM.

If all asbestos sources generated identical fibre size distributions, then measuring a fraction of that distribution would give us a perfect index of the total number of fibres, and therefore the health hazard, assuming still

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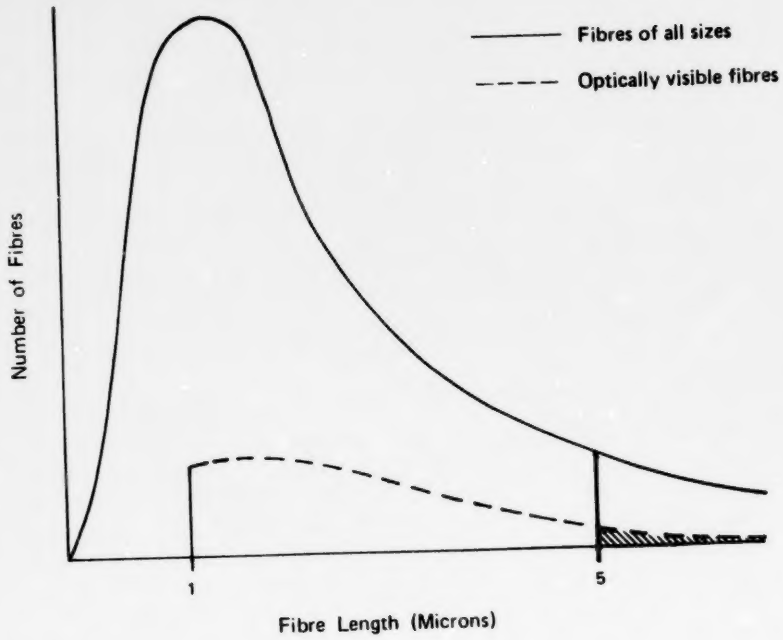
[33] Eric J. Chatfield, *Measurement of Asbestos Fibre Concentrations in Ambient Atmospheres*, Royal Commission on Asbestos Study Series, no. 10 (Toronto: Royal Commission on Asbestos, 1983), p. 13.

Figure 7.3

Relationship Between Total Fibre Distribution and Optical Fibre Count



Note: Shaded area shows optical fibre count.

SOURCE: Eric J. Chatfield, *Measurement of Asbestos Fibre Concentrations in Ambient Atmospheres*, Royal Commission on Asbestos Study Series, no. 10 (Toronto: Royal Commission on Asbestos, 1983), Figure 6, p. 15.

that all fibres are equally hazardous. There is, however, no reason to expect different dust sources to generate similar size distributions. Figure 7.4 shows that three sources might generate three very different distributions of fibre size, with source A producing primarily short fibres, while source C produces primarily long fibres. Suppose that a TEM, which can detect all fibres, were used to examine the three dust clouds depicted in Figure 7.4, applying the 5 micron length limit. The fibre count for source A would be very low, because only a few percent of its fibres are long enough to qualify for counting under the 5 micron length limit. The fibre count for source C, on the other hand, would be very large, since the vast majority of those fibres are longer than 5 microns. The fibre count for source B would be between that of A and C. Thus, three sources which generated identical total numbers of fibres would yield enormously different measured fibre counts under current counting rules that incorporate a 5 micron cutoff. If all fibres were of equal toxicity, the measurement only of longer fibres, or of fibres thick enough to be detected optically, provides us with an unreliable measurement that will yield varying fibre counts among different sources and that will not bear a consistent relationship to the total number of fibres.

Consider a second case in which toxicity varies by fibre size. In this case it is not desirable to count all fibres. Instead, we would wish to count only those fibres of high toxicity. To take the worst example, suppose that while only fibres shorter than 5 microns were counted, only fibres longer than 5 microns were toxic. Under these assumptions, source C would present a far greater hazard than source A, yet the fibre count at source A would be enormously greater than at C. Such a measurement method would be useless for distinguishing situations of different hazard levels. On the other hand, if only fibres longer than 5 microns are toxic, and only fibres longer than 5 microns are measured, then the measure will correlate perfectly with the health hazard.

Thus, ideally, the rules for determining which fibres should be counted should be based on medical evidence of the toxicity of the various fibres. Dr. Friedrich Pott hypothesized that toxicity was a function of fibre length and diameter, with low levels of toxicity below lengths of 5 microns, and high toxicity levels around lengths of 20 microns.[34] (See Figure 7.5.) In Chapter 5 we conclude that toxicity increases with fibre length, so that the most relevant fibres are those longer than 5 or perhaps 8 microns. We also conclude that thin fibres are likely to be more hazardous than thick fibres. These conclusions suggest that counting fibres longer than 5 microns is a much better index of the health hazard than would be a count of fibres of all lengths. While a somewhat greater length cutoff, such as 8 microns,

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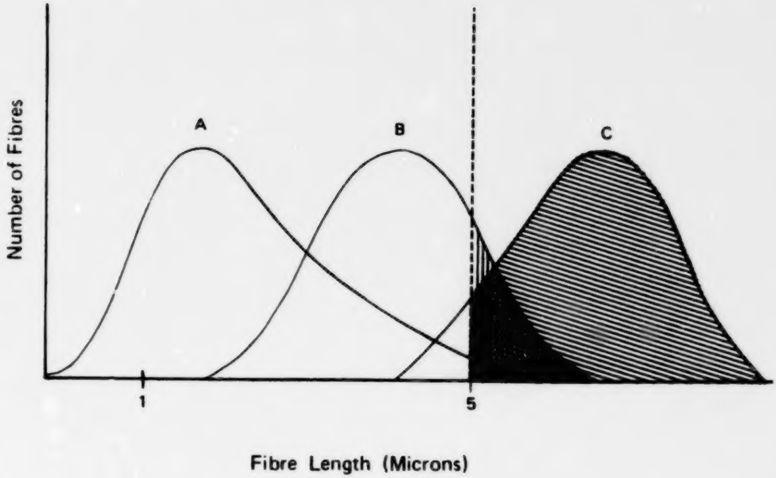
[34] Friedrich Pott, "Some Aspects on the Dosimetry of the Carcinogenic Potency of Asbestos and Other Fibrous Dusts," *Staub-Reinhalt. Luft* 38:12 (December 1978): 486–490.

Figure 7.4

Three Examples of Fibre Length Distributions



might be still better, the evidence on this is not sufficient to give us confidence in recommending any change from the current 5 micron size limit used in the Ministry of Labour's code for measuring fibres. It follows that the mass concentration of asbestos in the air is a less accurate predictor of health effects than would be a count of fibres over 5 microns, since there is no reliable relationship between mass and the count of long, thin fibres we believe to be of greatest medical concern. Our conclusions also suggest that a count of fibres over 5 microns made by a TEM will be a better predictor of health effects than one made by the optical microscope, since more thin fibres will be detected by the TEM.

Ideally, it would be desirable to know the relative toxicity of fibres of all sizes and to measure the concentration of fibres of all sizes. Such data would allow one to construct a health index for any air sample by multiplying toxicity of each fibre size by its concentration. Our general conclusion that long, thin fibres are of greatest concern, however, is not sufficiently precise to provide a toxicity index that would allow us to recommend such a procedure. While the TEM may be able to measure more accurately than the PCM the long, thin fibres that are most toxic, there are serious barriers to the use of the TEM for workplace measurement. There is as yet no standardized method for TEM measurement, so fibre counts are not reproducible among laboratories. The TEM costs at least 10 times as much as the PCM for standard workplace monitoring. We believe that the use of PCM, with a 5 micron minimum size for fibre counting, represents a reasonable compromise between measuring accurately the fibres of greatest health consequence, and restraining the cost of such measurement in the workplace. For environmental measurement, where the fibre concentration is generally far lower than in the workplace, we believe that TEM measurement is essential. (See Chapter 9.)

Because the length of maximum toxicity may be greater than 5 microns, it might be useful to provide, in addition to the count of fibres longer than 5 microns, a count of fibres longer than, for example, 15 microns. At present, however, we would not be able to make any particular use of such data. Furthermore, Dr. Chatfield has reported that if optical microscope operators were required to report separately the number of fibres longer than 20 microns, as well as the number longer than 5 microns, this would tend to reduce the reproduceability and accuracy of these results.[35] It is not, therefore, advisable to gather such additional length information routinely in workplace monitoring.

We have rejected the optical microscope for routine measurement of fibre size distribution because of low accuracy, and the TEM because of

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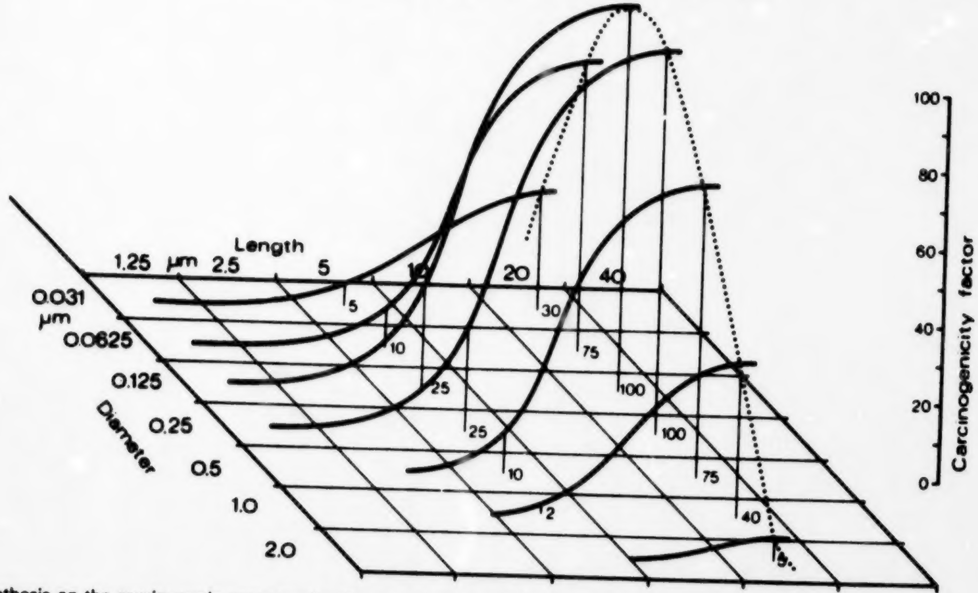
[35] Chatfield, *Measurement of Asbestos Fibre Concentrations in Workplace Atmospheres*, p. 55.

Figure 7.5

Hypothesized Carcinogenic Potency as a Function of Fibre Size*



Note: *Hypothesis on the carcinogenic potency of a fibre as a function of its size with some data on "carcinogenicity factors." (This three-dimensional model requires the fibre size of a sample to be divided into numerous categories. The size categories include three parameters: length, diameter, and the length/diameter ratio.)

SOURCE: Friedrich Pott, "Some Aspects on the Dosimetry of the Carcinogenic Potency of Asbestos and Other Fibrous Dusts," *Staub-Reinhalt. Luft* 38:12 (December 1978): 487 (Figure 3).

cost and the absence of standard methods. However, it is possible that the scanning electron microscope (SEM) can perform fibre counts at a cost little above that of an optical microscope, where fibre identification is not necessary. Dr. Chatfield has stated that the SEM can measure not only very thin fibres which are not visible in the optical microscope, but can also be used to report fibre length.[36] This method is not, however, proved for the enforcement of workplace control limits. It would be useful to develop better information on the size distributions of asbestos fibres in Ontario workplaces and to analyze the usefulness of the SEM as a workplace monitoring tool. We recommend that:

7.10 The Ministry of Labour should conduct research using a scanning electron microscope alongside an optical microscope to explore the size distribution of fibres found in various workplaces.

B.6 Measuring Fibre Type

Dr. Chatfield concluded that the membrane filter method is not suitable for measuring separately the concentrations of different types of asbestos in mixed fibre atmospheres.[37] The membrane filter method is satisfactory, however, for measuring the total fibre concentration in such atmospheres. The Regulation Respecting Asbestos specifies different control limits for different fibre types, but requires that the total fibre count meet the most strict applicable control limit. Thus, it is unnecessary under the Regulation Respecting Asbestos to perform separate counts of chrysotile, amosite, and crocidolite. Our recommendations in Section F of this chapter will endorse these distinctions among fibre types and the application of the total fibre count in a mixed atmosphere to the relevant control limit. Therefore, we see no need to develop more sophisticated measurement techniques which can distinguish among fibre types in the workplace for routine enforcement of control limits.

C. Estimates of Future Disease from Future Workplace Exposure

C.1 Introduction

In Chapter 5 we review the medical evidence on the health effects of exposure to asbestos. This includes biological evidence, epidemiological studies, lung tissue studies, and animal studies. We conclude in that chapter that the risk of respiratory disease from exposure to any specified airborne asbestos fibre concentration differs among the three major fibre types and differs among processes generating the dust. Determining the control limits that should govern worker exposure to asbestos, however, requires quantitative estimates of the magnitude of disease that might arise from the specification of certain control limits. In this section we will use the major epidemiological studies, and the models described in the Appendix to this chapter, to produce a quantitative risk assessment. This risk assessment will be supplemented in Section F of this chapter with other data and conclusions from Chapter 5 to form a basis for recommending appropriate regulatory responses.

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[36] Ibid., p. 57.

[37] Ibid., p. 63.

C.2 The U.K. Advisory Committee Predictions

The incidence of asbestos-related disease that may result from future exposure to asbestos in the workplace may be predicted using a dose-response model and data from past studies which relate exposure and disease rates. The U.K. Advisory Committee report attempted to summarize the mortality experience shown in eleven studies of the health of asbestos workers.[38] The Advisory Committee concluded that if a 2 f/cc standard were adopted and complied with, and if employees were exposed over a 50-year working life, then the asbestosis incidence might be as great as 0.5% per annum, or it might be considerably less.[39] The Advisory Committee further concluded that the excess mortality from lung cancer after a 50-year working life might range from 0.1 to 2%, with dust levels ranging from 0.02 to 11 f/cc, the range being attributable to differing assumptions and different studies used as the basis for the forecast.[40]

We conclude in Chapter 5 that at cumulative exposures of 5 fibres per cubic centimetre-years (f/cc-yrs) or as high as 25 f/cc-yrs, asbestosis will be a very rare disease and may even in fact become a disease of the past. Even at these low doses, some fibrosis is possible, but clinical manifestations are not likely to develop in an individual's lifetime. In addition, it is unlikely that any significant number of workers would be exposed to 1 f/cc throughout their entire working career, since many work stations in the Ontario asbestos manufacturing industry today already are below that fibre level most or all of the time.

We conclude that with the recently adopted workplace control limit of 1 f/cc for chrysotile, and less for the other types of asbestos, the risk of asbestosis will be sufficiently small that it can be disregarded in comparison with the risk of cancer, to which we now turn.

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[38] U.K., Advisory Committee on Asbestos, *Asbestos — Volume 1: Final Report of the Advisory Committee*, Table 13, p. 51.

[39] Ibid., paragraph 94, p. 55.

[40] Ibid., Table 21, p. 62.

C.3 Projecting Lung Cancer and Mesothelioma

(a) Worker Exposure Under Control Limits

Assessing the aggregate health effects of an occupational asbestos control limit requires making some assumptions about the worker and his exposure. We will define two cases for analysis: the short-term, normal exposure; and the long-term, high exposure.

As was pointed out in Section B of this chapter, it can be conservatively estimated that the average worker is exposed over the course of a year to an average of one-half the fibre concentration of the control limit if the control limit is enforced. If the control limit is not adequately enforced, then some workers may be exposed to more than one-half the control limit. We believe that it is reasonable to represent the exposure of a highly exposed worker over a year by a concentration equal to the control limit itself. The next step is to estimate the average duration of a worker's exposure to asbestos in his employment; that is, the number of years the worker is in asbestos manufacturing. At two Ontario brake plants, the average worker had been with the firm for about 10 years, although a third plant's workers averaged less than 4 years, and some workers had been at their plant for 25 years or more.[41] In testimony, we heard of a number of workers at the Scarborough plant of Johns-Manville who developed asbestos disease after 25 years of service.[42] There is little labour migration from one asbestos manufacturing plant to another, so time at a plant is a reasonable estimate of time in asbestos manufacturing. We will represent a short-term worker by an exposure duration of 10 years, and a long-term worker by an exposure duration of 25 years. While some workers may be exposed for more than 25 years, and others for less than 10 years, the vast majority of exposures should be within or below these limits. Coincidentally, our short-term exposure is exactly the minimum duration of exposure stipulated in the Ontario Workers' Compensation Board guideline for asbestos-related lung cancer claims.

Combining the exposure and the duration, the short-term, normally exposed worker would be exposed to a fibre concentration equal to one-half the control limit for a period of 10 years. The long-term, highly exposed worker would be exposed to a fibre concentration equal to the control limit for a period of 25 years. We do not believe that exposures greater than the control limit are likely, absent a complete breakdown in vigilance regarding the hazards of asbestos.

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[41] Luce and Swimmer, *Worker Attitudes About Health and Safety in Three Asbestos Brake Manufacturing Plants*, p. 4.2. A recent study of three U.S. asbestos manufacturing plants reported that the average tenure of workers was 8.7 years, 7.6 years, and 9.2 years. See Alison D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant," *British Journal of Industrial Medicine*, in press, 1984, p. 6.

[42] See RCA Transcript, Submission by the Asbestos Victims of Ontario, 16 February 1981, Volume no. 1, especially pp. 153, 156, 163, 167, 176.

We can assess the health effects of various control limits by applying the above conclusions about worker exposure to the dose-response estimates that have emerged from previous epidemiological studies. As a starting point, we will look at the effects of exposure to 2 f/cc for 50 years, which produces a cumulative exposure of 100 f/cc-yrs, a high exposure that has been referred to in many studies. We will also consider the exposure for a short-term, normally exposed worker resulting from a 1 f/cc control limit for 10 years, which yields a cumulative exposure of 5 f/cc-yrs. We finally consider the case of the long-term, high exposure worker under a 1 f/cc control limit for 25 years, which yields a cumulative exposure of 25 f/cc-yrs.

(b) Relative Risk Projections

We asked Mr. Ronald J. Daniels and Professor Robin S. Roberts to analyze for this Commission major studies of asbestos workers for which there are reasonable data on the fibre levels to which workers were exposed. Their analysis, "Predicting Workplace Health Risks," appears as the Appendix immediately following this chapter.[43] Their summary of the health effects found in these studies is presented in Table 7.1. This table shows the risk of lung cancer, gastrointestinal cancer, mesothelioma, and asbestosis revealed by the studies conducted by: J. Corbett McDonald et al. [hereafter the J.C. McDonald study]; Vivian L. Henderson and Philip E. Enterline [hereafter the Enterline study]; Julian Peto (et al.) [hereafter the Peto study]; John M. Dement et al. [hereafter the Dement study]; Geoffrey Berry and Muriel L. Newhouse [hereafter the Berry study]; Murray M. Finkelstein [hereafter the Finkelstein study]; and Irving J. Selikoff, E.

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[43] See Ronald J. Daniels and Robin S. Roberts, "Predicting Workplace Health Risks," Appendix to Chapter 7 of this Report.

Cuyler Hammond, and Herbert Seidman [hereafter the Selikoff study].[44] The bottom of Table 7.1 reports the lung cancer rate alone as a function of fibre levels, showing the relative risks detected by the various studies. The relative risk is the ratio of the number of observed deaths to the number of expected deaths. The excess risk is the relative risk minus 1.0, expressed as a percentage.

In the Appendix to Chapter 7, Daniels and Roberts present an estimate of the relative risk of lung cancer from specified fibre exposures based on the dose-response relationships estimated from Table 7.1. This is summarized in Table 7.2. Consider the relative risk predicted for an exposure of 100 f/cc-yrs, the bottom line in this table. The J.C. McDonald data predict a relative risk of 1.02 to 1.046, for an excess risk of 2.0 to 4.6%. The Enterline data predict a relative risk of 1.069, for an excess risk of 6.9%. The Berry data predict a relative risk of 1.058, for an excess risk of 5.8%. The most alarming statistics in the table are those from Dement and Finkelstein, both of which predict a relative risk of about 5.2, for an excess of 420% at 100 f/cc-yrs. It is obvious that the Dement and Finkelstein data predict enormously greater health risks from a given fibre exposure than do the other studies.

The coefficients and equation presented in Table 7.2 can be used to estimate the risk presented at the lower exposure levels experienced by our short-term, normally exposed or long-term, highly exposed workers. Under a 1 f/cc control limit a short-term, normally exposed worker would be exposed to 5 f/cc-yrs over his work life. The equation from Table 7.2 would predict an excess risk from lung cancer of less than 1% at this fibre level, with two exceptions: both the Dement and Finkelstein studies would predict

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[44] J. Corbett McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910–75," *British Journal of Industrial Medicine* 37 (1980): 11–24; J. Corbett McDonald et al., "Mortality in the Chrysotile Asbestos Mines and Mills of Quebec," *Archives of Environmental Health* 22 (June 1971): 677–685; Vivian L. Henderson and Philip E. Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 117–126; Julian Peto et al., "A Mortality Study Among Workers in an English Asbestos Factory," *British Journal of Industrial Medicine* 34 (1977): 169–173; Julian Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 829–836; John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Workers," *Annals of Occupational Hygiene* 26:1–4 (1982): 869–887; Geoffrey Berry and Muriel L. Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," *British Journal of Industrial Medicine* 40:1 (February 1983): 1–7; and Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983. (Mimeographed.) See also, Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943–1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 91–116.

Table 7.1
Mortality Experience in Seven Epidemiological Studies

Result	J.C. McDonald* (Mining)	Enterline (Manufacturing)	Peto (Textiles)	Dement (Textiles)	Berry*,** (Friction Products)	Finkelstein*** (Asbestos-Cement)	Selikoff (Insulation)
a) Overall risks (≥ 20 years since first exposure)	O E RR§	O E RR	O E RR	O E RR	O E RR	O E RR	O E RR
(i) Lung cancer	230 184.5 1.25	63 23.3 2.70	22 13.8 1.59 (Hired ≤ 1950) 8 1.62 4.94 (Hired ≥ 1951)	23 5.7 4.04	143 138.5 1.03†	21 4.1 5.12	450 93.7 4.8
(ii) Gastrointestinal cancer	209 203 1.03	55 45.6 1.21	16 15.7 1.02	9 7.10 1.27††	103 107.2 0.96	8 2.8 2.83	93 53.2 1.8
(iii) Pleural mesothelioma deaths	10	5	7	0	8		61
(iv) Peritoneal mesothelioma deaths	0	0	0	1	0	8 (15 best evidence)	108
(v) Fibrotic lung disease deaths	42	31	8 (Hired ≤ 1950) 0 (Hired ≥ 1951)	15††	Not mentioned	9 (non malignant respiratory disease)	204 53.8 3.8
b) Lung cancer by dust/fibre level	mppcf-yrs RR < 10 1.0 10-99 1.2 100-299 1.2 300-599 1.8 ≥ 600 2.9	mppcf-yrs RR < 125 1.98 125-249 1.80 250-499 3.28 500-749 4.50 ≥ 750 7.78	Not reported	f/cc-yrs RR approx. mppcf-yrs < 27.4 2.23 < 9.1 27.5-108.5 3.57 9.1-36.5 108.6-273 9.78 36.5-91.3 ≥ 274 15.53 91.3-182.6	f/cc-yrs† RR 50 1.03 100 1.06 150 1.09	f/cc-yrs RR < 30 2.31 30-75 6.15 75-105 12.08 105-150 9.0 ≥ 150 2.69	Not reported

Notes: *These data are for the men only, not the entire cohort.
The J.C. McDonald cohort included 10,939 men (3,291 dead), and the Berry cohort included 9,113 men (1,787 dead).
**Ten years since first exposure.
***Production workers only.
§"O" means observed; "E" means expected; and "RR" means relative risk.
†Simulated exposure.
††Includes all deaths from first exposure.

SOURCE: Appendix to Chapter 7, Tables 6, 7.

Table 7.2

Estimated Relative Risk Coefficients for Lung Cancer

	J.C. McDonald (Mining)		Enterline (Manufacturing)	Peto (Textiles)	Dement (Textiles)	Berry (Friction Products)	Finkelstein (Asbestos-Cement)	Selikoff (Insulation)
Coefficient Value C*	**at 3 f/cc** 0.000463 **at 7 f/cc** 0.000198		**at 9.5 f/cc** 0.000693	0.01	**at 3 and 8 f/cc** 0.0416	0.00058	0.042	0.0101
Estimated Risk	**3 f/cc**	**7 f/cc**						
25 f/cc - yrs	1.012	1.005	1.017	1.25	2.04	1.015	2.05	1.25
50 f/cc - yrs	1.023	1.010	1.035	1.5	3.08	1.029	3.1	1.50
75 f/cc - yrs	1.035	1.015	1.052	1.75	4.12	1.044	4.15	1.76
100 f/cc - yrs	1.046	1.020	1.069	2.0	5.16	1.058	5.2	2.01

Note: *The coefficient "C" may be inserted in the equation RR = 1 + C x FCC x D to estimate the relative risk of lung cancer from exposure to an asbestos concentration of FCC for a duration of D years.

SOURCE: Appendix to Chapter 7, Table 8.

a 21% excess risk. We are faced, therefore, with epidemiological studies that differ by more than a factor of 100 in the amount of excess lung cancer risk that they predict from particular exposures to asbestos dust. The long-term, highly exposed worker would be exposed to 25 f/cc-yrs over his work life. Table 7.2 shows excess lung cancer risks at this exposure level of less than 2% for J.C. McDonald, Enterline, and Berry and 25% for Peto and Selikoff, with Dement predicting a 104% excess risk and Finkelstein, a 105% excess risk.

(c) Absolute Risk and Projections

The Appendix to Chapter 7 estimates the magnitude of health effects arising from different exposure levels.[45] It estimates the risk of death and the number of person-years of life lost from death resulting from lung cancer and mesothelioma at several fibre levels, relying on seven epidemiological studies. The Selikoff study included no exposure data, so the dose-response relationship was calculated using exposure estimates derived indirectly by the U.S. Environmental Protection Agency (EPA) based on dust measurements in other places at other times. The Appendix calculates effects based on the dose-response relationship of each study, using a detailed calculation of the number of workers surviving to each age, a more detailed procedure than that used in Table 7.2. The model is one in which the lung cancer incidence is proportional to the intensity (f/cc) and duration of asbestos exposure, with a delay period of 10 years after onset of exposure.[46] The incidence of mesothelioma, using a very different model, is proportional to the intensity of exposure and is related to the fourth power of both time since first exposure and duration of exposure.[47] The Appendix assumes a hypothetical workforce of 1,000 persons who would work for 10 years beginning at age 22 and simulates the number of deaths that would result from various fibre exposures. A 25-year duration of exposure is also considered. The results of these simulations are shown in Table 7.3. There is again a wide range of predicted mortalities, with the analysis based upon the Dement and the Finkelstein data showing the greatest risks and those based upon the J.C. McDonald and the Berry data showing the least. On average in this simulation, those who do die will lose an average of approximately 13 years of life. Thus, the deaths occur at a relatively late age, as one expects with cancer. The combined mortality is slightly less than proportional to the fibre level in all studies, so that reducing the fibre level from 2 to 1 f/cc divides the death rate almost in half, and reducing it to 0.5 f/cc divides it in half again. Unlike Table 7.2, Table 7.3 includes mesothelioma deaths. Although the Berry cohort experienced 8 deaths from mesothelioma, more than the number of excess lung cancer deaths, this simulation includes lung cancer only, because Berry attributed the mesothelioma deaths to crocidolite.

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[45] See the Appendix to Chapter 7 of this Report.

[46] For details of the model, see the Appendix to Chapter 7. The essence of the model is that the number of excess deaths per year at age a in a cohort of size NOW first exposed to asbestos at age A is given by the following equation:

IE(a) = C*FCC*D*NOW*LU(a)*S(a)/S(A),

Where:

IE(a) = number of excess lung cancer deaths per year at age a resulting from asbestos exposure beginning at age A;

C = a proportionality constant derived from epidemiological studies;

FCC = average intensity of exposure to asbestos in f/cc;

D = duration of exposure to asbestos;

NOW = number of workers exposed;

LU(a) = the age-, gender-, and smoking-specific death rate for lung cancer;

S(a), S(A) = the cumulative probability of survival to ages (a) and (A) respectively.

These calculations estimate the risk from a short-term, normal exposure for 5 f/cc-yrs, which is shown in the 10-year column and the 0.5 f/cc exposure levels. The J.C. McDonald, Enterline, and Berry data would predict asbestos-related cancer mortality rates of considerably less than one death per 1,000 exposed workers at this exposure level. The Selikoff and Peto data would predict between 6.2 and 8.2 asbestos-related cancer deaths per 1,000 exposed workers. The Dement data predict 16 deaths, while the Finkelstein data predict a staggering 82 deaths, over 8% of the workforce. The Finkelstein data reveal cancer risks hundreds of times greater than those of some other studies at a given measured asbestos fibre concentration. It is possible that the model used here has overestimated the mesothelioma deaths that may occur in the future, especially in the Finkelstein cohort with its high mesothelioma rate, because of the assumption that mesothelioma deaths will continue to rise rapidly as the cohort ages. This assumption cannot be tested until the passage of time reveals the actual disease incidence of this cohort. Even if the assumption is unfounded, the

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[47] $MI(a) = K \cdot [T^4 - (T-D)^4] \cdot FCC \cdot NOW \cdot S(a)/S(A)$,

Where:

MI(a) = number of mesothelioma deaths per year at age a in a cohort of size NOW resulting from asbestos exposure beginning at age A;

K = a proportionality constant derived from epidemiological studies;

T = time since first exposure = a - A;

D = duration of exposure to asbestos;

FCC = fibre intensity in f/cc;

NOW = number of workers exposed;

S(a), S(A) = cumulative probability of survival to ages (a) and (A) respectively.

Table 7.3

Estimated Lifetime Mesothelioma and Lung Cancer Mortality Rates per 1,000 Asbestos-Exposed Workers*

Epidemiological Study	Fibre Exposure Level	10-Year Exposure Mortality	Total Life-Years Lost	25-Year Exposure Mortality
Selikoff (assumed historical exposure = 15 f/cc)	2.0	32	431	64
	1.0	16	217	32
	0.5	8.2	109	16
	0.2	3.3	44	6.6
Peto	2.0	25	321	52
	1.0	12	161	26
	0.5	6.2	81	13
	0.2	2.5	32	5.3
McDonald 1: (assuming 3 f/cc = 1 mppcf)	2.0	0.7	9	1.8
	1.0	0.4	4.5	0.9
	0.5	0.2	2.3	0.5
	0.2	0.1	0.9	0.2
McDonald 2: (assuming 7 f/cc = 1 mppcf)	2.0	0.3	3.9	0.8
	1.0	0.2	1.9	0.4
	0.5	0.1	1.0	0.2
	0.2	0.03	0.4	0.1
Dement	2.0	62	782	144
	1.0	32	398	76
	0.5	16	201	39
	0.2	6.5	81	16
Enterline	2.0	1.1	14	2.7
	1.0	0.5	6.7	1.4
	0.5	0.3	3.4	0.7
	0.2	0.1	1.4	0.3
Finkelstein	2.0	295	4,193	460
	1.0	159	2,208	260
	0.5	82	1,133	138
	0.2	34	461	57
Berry	2.0	0.9	11	2.3
	1.0	0.5	5.6	1.1
	0.5	0.2	2.8	0.6
	0.2	0.1	1.1	0.2

Note: *Assumptions: Duration of exposure = 10 years; 25 years. Age at first exposure = 22 years.

SOURCE: Adapted from: Appendix to Chapter 7, Tables 10, 11.

Finkelstein data would predict overall mortality risks at least twice as great as the Dement study.

The lifetime long-term, high exposure risk for 25 f/cc-yrs of exposure may be read from the 25-year column and the 1 f/cc exposure levels. In this case, the J.C. McDonald, Enterline, and Berry data would predict asbestos-related cancer mortality rates of less than 2 deaths per 1,000 workers. The Selikoff and Peto data would predict between 26 and 32 asbestos-related cancer deaths per 1,000 workers. The Dement data predict 76 asbestos-related cancer deaths, almost 8% of the workforce; while the Finkelstein data predict 260 deaths, or over one-quarter of the workforce.

(d) OSHA Projections

Another estimate of the risk of cancer from asbestos exposure was made for OSHA by Han K. Kang and Kenneth Chu, called "Preliminary Risk Assessment for Asbestos." This report is still in draft form two years after it was first issued, but has recently been made public in the United States.[48] The authors reviewed five studies, adding the study by Herbert Seidman, Irving J. Selikoff, and E. Cuyler Hammond [hereafter the Seidman study] to those we have considered.[49] Their estimate of deaths is summarized in Table 7.4 for the exposure of 1,000 workers to 2.0, 0.5, or 0.1 f/cc for a 45-year work life. There is, however, reason to believe that the Kang and Chu mortality rates were overestimated in the case of the predictions based upon the Seidman study, which is Study #3 in Table 7.4. Kang and Chu estimated the exposure of the workers in the Seidman study based on dust measurements at other plants at other times to an average of about 35 f/cc for brief periods of time up to 2 years. At the time of the study, this intense but brief exposure had caused excess cancer mortality of about 15% of the workforce. When Kang and Chu used this experience to predict the health effects of exposure to 2 f/cc for 45 years, they assumed that a short, intense exposure is equivalent to a longer, less intense exposure, a proposition which we have questioned in Chapter 5. They predicted that over half of the workers exposed to 2 f/cc for their working life would die of asbestos-related cancer. Yet studies such as that by E. Cuyler Hammond, Irving J. Selikoff, and Herbert Seidman [hereafter the Hammond study] of other populations exposed to asbestos insulation for much longer periods show far lower disease rates than this.[50] Because the Seidman study was based on intense, short-term exposures, we believe it is incorrect to use the Seidman results for assessing the risk from exposure to 2 f/cc or lower levels over a long period of time.

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[48] Han K. Kang and Kenneth Chu, "Preliminary Risk Assessment for Asbestos," draft report prepared for the U.S. Department of Labor, Occupational Safety and Health Administration, Washington, D.C., 8 December 1981. Reference to this risk assessment was made in the U.S. Court of Appeals during litigation on enthylene oxide. This case was reported in *Public Citizen Health Research Group, et al. v. Thorne G. Auchter, Assistant Secretary, Occupational Safety and Health Administration, et al., Appellants* 702 F.2d 1150 (1983). While this Commission's Report was in press, the U.S. Occupational Safety and Health Administration published a quantitative risk assessment in connection with its notice of an Emergency Temporary Standard on November 4, 1983. See U.S., Department of Labor, Occupational Safety and Health Administration, "Occupational Exposure to Asbestos; Emergency Temporary Standard," 29 CFR Part 1910, 48 FR 51086-51140, 4 November 1983. This Emergency Temporary Standard was stayed by court order. Comparing the dose-response coefficients reported in Table 9 of that notice with those reported in the Appendix to the present chapter, we find that of 10 comparable coefficients, in 3 cases the OSHA coefficients are virtually identical to ours, in 4 cases the OSHA coefficients are within 50% of ours, and 2 coefficients differ by up to a factor of 2. One coefficient, that for the Enterline study, differs by considerably more because of differing assumptions about the conversion of particle counts to fibre concentrations.

[49] See Herbert Seidman, Irving J. Selikoff, and E. Cuyler Hammond, "Short-Term Asbestos Work Exposure and Long-Term Observation," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 61–89.

The other total risk estimates presented by Kang and Chu were greater than those of Daniels and Roberts in the Appendix to this chapter, primarily because Kang and Chu assumed a longer duration of exposure, and for some studies Kang and Chu used a different conversion rate between particles and fibres.

(e) Analysis of the Projections

Are the risk assessments made by Daniels and Roberts in the Appendix to this chapter and by Kang and Chu consistent or inconsistent? Kang and Chu differ in part because they assumed a longer duration of exposure. We can, however, use the models in the Appendix to calculate risks for exposures similar to those assumed by Kang and Chu. Inserting in the equations from Table 7.2 an exposure of 45 years at 0.5 f/cc or 22.5 f/cc-yrs, one of the exposures used by Kang and Chu, and assuming that the average lung cancer risk for a non-asbestos exposed worker is 0.075, we can calculate the excess cancer mortality per 1,000 workers as shown in Table 7.5. The model of Table 7.3 can also be used to calculate risks for 0.5 f/cc for 45 years as shown in Table 7.5. As Table 7.5 shows, the three risk assessments produce very similar risks when similar exposures are assumed, for the studies covered by all analysts. For the J.C. McDonald study, the highest estimate of risk is virtually the same. The Dement estimates are also within 15% of each other. The Kang and Chu estimates based on Enterline are 2 or more times those of the Appendix to Chapter 7, which is entirely the result of different conversion rates between particles and fibres.

While we have shown that the risk assessment by Kang and Chu, except for their use of the Seidman study, is not inconsistent with the

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[50] E. Cuyler Hammond, Irving J. Selikoff, and Herbert Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 473–490.

Table 7.4

OSHA Draft Cancer Risk Estimates
(Lifetime Cancer Risk for 1,000 Workers After 45-Year Exposure)*

Exposure Level	2 f/cc	0.5 f/cc	0.1 f/cc
Excess lung cancer			
Study #1**	8-43	2-11	0.4-2
2	253-260	71-75	14-15
3	467	145	32
4	70	18	3.6
5	3	0.75	0.2
Excess total cancer			
Study #1	13-70	3-18	1-4
2	***	***	***
3	632	208	48
4	128	34	7
5	***	***	***

Notes: *All estimates based on linear dose-response model. Other models yield similar or lower estimates.

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Study #1 Vivian L. Henderson and Philip E. Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 117-126. For Study #1, the low estimate assumed that 1 mppcf = 5 f/cc, while the high estimate assumed that 1 mppcf = 1 f/cc.

Study #2 John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," Doctoral dissertation, University of North Carolina, Chapel Hill, North Carolina, 1980. For Study #2, the assumed historic exposure is up to 91 mppcf.

Study #3 Herbert Seidman, Irving J. Selikoff, and E. Cuyler Hammond, "Short-Term Asbestos Work Exposure and Long-Term Observation," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 61-89. For Study #3, the assumed historic exposure is up to 52.5 mppcf-yrs.

Study #4 E. Cuyler Hammond, Irving J. Selikoff, and Herbert Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 473-490. For Study #4, the assumed historic exposure is up to 15 f/cc.

Study #5 J. Corbett McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," *British Journal of Industrial Medicine* 37 (1980): 11-24. For Study #5, the assumed historic exposure is often over 300 mppcf-yrs.

***Data not available.

SOURCE: Adapted from: Han K. Kang and Kenneth Chu, "Preliminary Risk Assessment for Asbestos," draft report prepared for the U.S. Department of Labor, Occupational Safety and Health Administration, Washington, D.C., 8 December 1981.

Table 7.5

Comparison of Risk Assessments

Epidemiological Study		**Projected Asbestos-Related Cancer Mortality per 1,000 Workers Exposed to 22.5 f/cc-yrs**		
		Derived from Table 7.3	Derived from Table 7.2	Kang and Chu
J.C. McDonald	1	0.76	0.75	0.75
	2	0.33	0.38	
Dement		65	71	71-75
Enterline		1.1	1.2	2-11

Appendix to Chapter 7 for a given fibre exposure, we consider that the 45-year exposure used by Kang and Chu is not a representative basis for risk assessment. We indicate above that we believe that a 10-year exposure and a 25-year exposure can represent the vast majority of exposures faced by workers in Ontario. We will therefore use the risk estimates of Table 7.3 for the short-term, normal exposure and the long-term, high exposure as a basis for evaluating control limits.

What should be done with the disparate risks suggested by the various epidemiological studies? We could assume that all are measuring the same risk and combine them. The Appendix to Chapter 7 evaluates the methodology of these studies and concludes that all are reasonably robust, and that none of them can be rejected on methodological grounds. These studies represent attempts to estimate the actual relationship between asbestos exposure and disease, with each estimate subject to some uncertainty or error. No one estimate is necessarily correct, but all might be used together to derive a better estimate than any alone. The average risk of the Table 7.3 studies, weighted by the number of subjects in each cohort, is about 5 asbestos-related deaths per 1,000 workers at an exposure of 5 f/cc-yrs. In a weighted average, the large Selikoff, Berry, and J.C. McDonald studies exert a considerable influence on the result, diluting the high risk estimates from the Dement and Finkelstein data.

However, we believe that the studies are measuring different risks. In Chapter 5 we differentiate risks by fibre type. We conclude that historical exposures to crocidolite and amosite were associated with higher disease rates than exposure to chrysotile. We conclude that mesothelioma is generally associated with exposure to amosite or crocidolite, and rarely with exposure to chrysotile. We recognize that chrysotile creates less dust in general, and further conclude that it is less hazardous at a given measured fibre level. These differences arise in part because the toxicity of fibres depends on their dimensions, and at a given optical count of fibres longer than 5 microns, amosite and crocidolite dust clouds contain a higher proportion of the long, thin fibres that are most dangerous than does a chrysotile dust cloud. Therefore, we reject the use of studies of crocidolite and amosite exposure to predict hazards from exposure to chrysotile.

We also conclude in Chapter 5 that for a single fibre type such as chrysotile, the health risk depends upon the process causing fibre release. We conclude that a given measured fibre concentration in mining and in brake manufacturing causes far less disease than the same measured fibre level in textile spinning and weaving. Working with sprayed insulation and pipe and boiler insulation causes intermediate risk levels. These differences arise in part because the toxicity of fibres depends on their dimensions and because the airborne fibre size distribution differs among processes. Thus, we reject combining studies of these processes to yield a single dose-response relationship.

(f) Summary of Projections by Fibre Type and Process

Our conclusion in Chapter 5 that fibre type and process may affect the risk to workers requires us to separate the studies discussed here for purposes of risk assessment. We will consider separately chrysotile atmospheres, and mixed atmospheres containing chrysotile and either amosite or crocidolite or all three. We will also separate mining, two general types of manufacturing, and other processes. In each case, we will identify the studies that we believe are relevant for risk assessment and determine the risk presented by exposure under whichever of the recently adopted Ontario control limits is applicable. The results are summarized in Table 7.6.

Mining — Chrysotile — In Ontario, natural deposits of asbestos appear to include only chrysotile, with no amosite or crocidolite.[51] This is true for asbestos mines and for other mines in which asbestos may appear as a contaminant. The J.C. McDonald study is the only study of the health effects of chrysotile miners. Therefore, we will use the J.C. McDonald study to estimate the risk from mining exposures to chrysotile in Ontario. The recently adopted Ontario control limit for chrysotile exposure is 1 f/cc. Table 7.6 shows that the short-term, normal exposure under this control limit yields a risk of 0.1 to 0.2 deaths per 1,000 workers; while a long-term, high exposure yields a risk of 0.4 to 0.9 deaths per 1,000 workers.

Manufacturing — Chrysotile Only — Ontario manufacturers of asbestos products today use, as an input, chrysotile fibres exclusively, to the best of our knowledge. However, not all manufacturing processes are alike. We will distinguish two general types of manufacturing processes for risk assessment purposes. In the first process, chrysotile fibres are mixed with binders and formed into a hard product such as a brake lining, gasket, or floor tile. In this process, little work is performed on the asbestos fibres before they are submerged in materials which will tend to trap these fibres. Subsequent work on the material, such as grinding, will tend to reduce fibre lengths, perhaps below the hazardous range.

The second type of process is one in which extensive work is performed on the fibres without encapsulating them in some other material. In a textile plant, for example, asbestos fibres are woven into yarn and then spun into cloth. Although in some plants today water is used to reduce fibre release, in this process, in general, there is considerable work on the fibre while it is in relatively free form. We will use a textile plant as a typical example of the second type of process.

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[51] For an analysis of natural deposits of magnesite, talc, and asbestos in Ontario, see Ulrich Kretschmar and Dianne Kretschmar, *Talc, Magnesite and Asbestos Deposits in the Kirkland Lake - Timmins Area, Districts of Timiskaming and Cochrane*, Ontario Geological Survey Open File Report 5391 (Toronto: Ontario Ministry of Natural Resources, 1982).

Table 7.6

Risk Assessment by Process and Fibre Type Recent Ontario Control Limit

Process	Fibre Type	Study Used	Applicable Control Limit (f/cc)	Mortality Risk per 1,000 Workers at the Control Limit	
				Short-term, Normal Exposure*	Long-term, High Exposure**
Mining	Chrysotile	J.C. McDonald	1.0	0.1-0.2	0.4-0.9
Manufacturing	Chrysotile				
a) General (friction products)		Berry	1.0	0.2	1.1
b) Textiles		Dement	1.0	16	76
		Peto***	1.0	6.2	26
Manufacturing	Mixed (crocidolite)	Enterline	0.2	0.06	0.3
		Peto***	0.2	1.3	5.3
		Finkelstein	0.2	17	57
Other (insulation work)	Mixed (amosite)	Selikoff	0.5	4.1	16

Notes: *Assuming a 10-year average duration of exposure at one-half the control limit.
**Assuming a 25-year duration of exposure at the control limit.
***There is uncertainty whether this textile plant used crocidolite as well as chrysotile.

SOURCE: Derived from Table 7.3.

The first type of process includes brake shoe manufacturing, which employs the vast majority of manufacturing workers exposed to asbestos in Ontario today. The Berry study of health effects at a brake manufacturing plant is therefore directly relevant to this sort of exposure. The Ontario control limit applicable to chrysotile manufacturing is 1 f/cc. Table 7.6 shows that the Berry study would predict 0.2 deaths per 1,000 workers for a short-term, normal exposure; and 1.1 for a long-term, high exposure.

The second type of manufacturing process is typified by a textile plant. Once again, the 1 f/cc control limit would apply. The Dement study of a chrysotile textile plant would predict 16 deaths per 1,000 exposed workers for a short-term, normal exposure; and 76 deaths for a long-term, high exposure.[52] The Peto study of a textile plant in the United Kingdom is the subject of some debate as to whether crocidolite was used in addition to chrysotile. If the Peto study should be treated as a pure chrysotile exposure, it would predict 6.2 deaths per 1,000 workers for a short-term, normal exposure; or 26 deaths for a long-term, high exposure.

Is this separation of processes, for purposes of risk assessment, inevitable or even plausible? It is plausible because the processes are very different. In a textile plant, the raw fibre is worked on in loose form in ways that could easily break fibre bundles into smaller bundles or individual fibres. In a brake plant, the rigid moulded brake lining is drilled and ground. Since the fibres are embedded in a hard material, such operations will tend to break fibres into *shorter* fibres as well as breaking them apart. This could easily lead to a higher proportion of short fibres, below the most toxic lengths. In this connection, Dr. Dement stated that "Compared with other operations using chrysotile asbestos, such as the friction and cement product industries, textile operations were found to have a substantially higher fraction of long-thin fibres which [Mearl F.] Stanton and others have implicated as important for development of respiratory cancer."[53]

The separation of processes is not only plausible, it is compelling because of the epidemiological studies. The risks derived from Dement are almost 100 times greater than those derived from Berry. It is extremely unlikely that such differences are the result of errors of methodology in the studies or chance variations in lung cancer mortality. If these results are accepted at face value, then *something* has caused different risks at these plants. In the absence of other explanations, we believe that the differences are caused by differences in the manufacturing process that yield, among other things, different fibre sizes.

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[52] The plant studied by Dr. Dement performed the following operations: preparation/waste recovery, carding, spinning, winding, twisting, and weaving. See Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," p. 876.

[53] Ibid., p. 883. (Discussion.)

Manufacturing — Mixed Fibres — In the past, Ontario manufacturing operations involving mixed fibres included the production of asbestos-cement pipe and the production of insulation products. While these processes no longer operate today, it is still important to predict the health risks that might occur if they were to resume. Dr. Enterline studied workers who produced asbestos-cement products, insulation, and textiles, and who were exposed to all three major types of asbestos. The relevant Ontario control limit would be 0.2 f/cc and the predicted mortality risk, less than 0.1 per 1,000 workers for a short-term, normal exposure, or 0.3 deaths for a long-term, high exposure. If the textile plant studied by Peto is regarded as including crocidolite, then it too is relevant for manufacturing with mixed fibres. At the 0.2 fibre control limit, 1.3 deaths for a short-term, normal exposure, or 5.3 deaths for a long-term, high exposure per 1,000 workers would be predicted. Finally, the Finkelstein study involved crocidolite and chrysotile exposure in the pipe plant. Applying the control limit of 0.2 f/cc, Table 7.6 shows that 17 deaths per 1,000 exposed workers might be anticipated from a short-term, normal exposure, or 57 deaths from a long-term, high exposure. We, therefore, have a considerable range of risk estimates for manufacturing operations in mixed fibre atmospheres.

Other Work — Mixed Fibres — A major category of work not so far covered is the installation and removal of asbestos-containing insulation in buildings and on pipes and boilers. The Selikoff study of insulation workers is directly relevant. Dr. Selikoff's workers were exposed to amosite as well as chrysotile, so the relevant Ontario control limit would be 0.5 f/cc. Table 7.6 shows that the risk at a 0.5 fibre control limit level would be 4.1 deaths per 1,000 workers for a short-term, normal exposure, and 16 deaths from a long-term, high exposure. In Chapter 9 we discuss the risk estimates produced by the Hammond study. If, as the U.S. EPA stated, the Hammond study found 59 excess deaths per 1,000 workers attributable to an exposure of, on average, 9 f/cc,[54] then exposure to an average of 0.5 f/cc would cause less than 2.5 deaths per 1,000 exposed workers.

Another form of exposure is field work on asbestos-cement products. No studies are available to shed any light on the risks from such work. An important question which we cannot resolve is whether there is any reason to expect that the risks from such work would be similar to those experienced in the manufacture of asbestos-cement products. As the Finkelstein study showed, the manufacturing risks are high indeed.

We have now established a basis for estimating the risk that may be expected in the primary exposures that Ontario workers may face in the future. It must be remembered that all of these estimates are subject to the considerable uncertainty inherent in the underlying epidemiological studies and the extrapolation of high exposure health experience to the effects of low exposures. We believe, however, that these estimates are the best that can be made with the available data. We will use these estimates in our derivation of the appropriate level for control limits later in this chapter. We return now to several subsidiary issues: the impact on asbestos-related cancer risks of worker turnover, worker age, and smoking.

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[54] U.S., Environmental Protection Agency, Office of Toxic Substances, *Support Document for Proposed Rule on Friable Asbestos-Containing Materials in School Buildings: Health Effects and Magnitude of Exposure*, EPA 560/12-80-003 (Washington, D.C.: U.S. Environmental Protection Agency, October 1980), Table 17, p. 80.

(g) Effects of Other Factors on Health Risks

Worker Turnover — The Appendix to this chapter explores the impact of worker turnover on the mortality rate from lung cancer and mesothelioma. This is of interest because of the Peto hypothesis that for mesothelioma incidence the duration of exposure is less important than the number of years since the first exposure to asbestos. Mr. Peto has argued, with growing support from others, including Dr. Finkelstein and Dr. Nicholson et al., that the intensity of earlier exposure is more important than the intensity of later exposure in determining mesothelioma disease rates.[55] He captured this in a model in which disease was determined by the intensity of exposure and the time since first exposure. In such a model, the level of exposure in later years is less important in determining disease rates. The results of the analysis in the Appendix to this chapter, using data from the studies listed in Table 7.3, are shown in Table 7.7. The third column in this table shows the total mortality in an industry where at any given time 1,000 employees are working, and where they are exposed to 1 f/cc, with a total of 20 years of activity in the industry. If each worker works only 4 years, then there would be five workforces employed during the 20-year period. The Peto data would predict 27 deaths resulting from this high turnover rate. If each worker stayed for 10 years, there would be two different workforces, and a total of 25 deaths. If a single group worked for 20 years, the death rate would drop to 22, a reduction of about 19%. The Peto hypothesis predicts that only mesothelioma deaths will be affected by worker turnover. The Dement data report virtually no mesothelioma deaths but many lung cancer deaths. Thus, changing worker turnover has a negligible impact on the overall mortality using the Dement data, as shown in Table 7.7. The Finkelstein data include a large number of mesothelioma deaths and produce a mortality reduction of 40% when turnover is reduced. If we accept Peto's model, then the mortality can be cut by up to 40% in situations where considerable mesothelioma is expected by reducing worker turnover to increase the average duration of employment from 4 to 20 years. However, no such effect will occur if that mortality arises only from lung cancer rather than mesothelioma.

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[55] See, for example, Julian Peto, "Dose and Time Relationships for Lung Cancer and Mesothelioma in Relation to Smoking and Asbestos Exposure," in *Zur Beurteilung der Krebsgefahren durch Asbest* [Proceedings of Bundesgesundheitsamt Asbestos Symposium], Berlin: February 1982, bga Shriften, MMV Medizin Verlag München, in press, 1983; Julian Peto, "An Alternative Approach for the Risk Assessment of Asbestos in Schools," report to the U.S. Environmental Protection Agency, 6 April 1981. (Mimeographed.) See also, Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory"; and William J. Nicholson et al., "Cancer from Occupational Asbestos Exposure: Projections 1980–2000," in *Banbury Report 9: Quantification of Occupational Cancer*, eds. Richard Peto and Marvin Schneiderman ([Cold Spring Harbor, New York]: Cold Spring Harbor Laboratory, 1981), pp. 87–111.

Age at First Exposure — Would there be any reduction in the risk of cancer mortality from asbestos exposure if the age at first exposure were increased? Suppose that the age at first exposure were 52 rather than 22 years. If there is a long latency period for cancers, then the 52 year old worker is unlikely to develop cancer before dying of natural causes, so the asbestos-related cancer risk would be greatly reduced. In the Peto model, the lung cancer risk is a function of age, while mesothelioma depends upon time since first exposure. In this model, later first exposure reduces the mesothelioma risk, while the lung cancer risk is reduced much less or even increased very slightly. The Daniels and Roberts Appendix calculates that raising the age of first exposure from 22 to 52, using their version of the Peto model, yields a 40% reduction in the total cancer risk using the Selikoff data and a 9% reduction using the Dement data, in which the risk is almost entirely from lung cancer rather than mesothelioma. Thus, increasing the age of first exposure to asbestos will in general reduce the cancer risk, although the reduction *may* be small where there is no risk of mesothelioma.

Smoking — What is the impact of smoking patterns on the risk of lung cancer and mesothelioma for asbestos workers? We observe in Chapter 5 that there is a strong multiplicative interaction between smoking and lung cancer, with heavy smokers in one study experiencing a lung cancer risk perhaps 11 times as great as that of non-smokers for a given level of asbestos exposure. We also conclude that smoking has no effect on the risk of mesothelioma. Thus, if workplaces that exposed workers to asbestos fibres employed only non-smokers, rather than the substantial proportion of smokers that are currently found, the overall cancer risk could be substantially reduced, especially in situations where the risk of mesothelioma is relatively small. The Appendix to this chapter analyzes the impact of replacing a plant workforce of 50% smokers with one consisting entirely of non-smokers, using the Peto model. Applied to the Dement data, the mortality risk reduction is 77%, because of the predominance of lung cancer in the Dement cohort. Using the Peto data, in which there are roughly similar proportions of lung cancer and mesothelioma, the risk reduction is 44%. It is therefore clear that employing only non-smokers can greatly reduce cancer risks from asbestos exposure where mesothelioma is not a major source of that risk. This is of particular importance in Ontario where,

Table 7.7

The Predicted Effects of Decreasing Worker Turnover
(2 f/cc; Age = 22)

Duration of Employment	Data Used	Total Mortality in One Workforce	Number of Workforces (X) Over 20 Years	Mortality for X Workforces to Receive 20 Years' Exposure
4 Years	Peto	5.4	5	27
	Dement	12.9		65
	Finkelstein	78.1		390
10 Years	Peto	12.4	2	25
	Dement	31.8		64
	Finkelstein	159		317
20 Years	Peto	22	1	22
	Dement	62		62
	Finkelstein	236		236

SOURCE: Adapted from: Appendix to Chapter 7, Table 14 (excerpt).

because crocidolite is no longer used, the risk of mesothelioma is far below that of lung cancer.

D. Current Fixed Workplace Asbestos Control Limits

Chapter 3 of this Report contains a detailed history of the regulation of worker exposure to asbestos fibres in Ontario, along with the relevant developments in the United States and the United Kingdom. As a background to our recommendations for control limits for such exposure, we present here a brief summary of the current status of the control limits in Ontario, other Canadian provinces, the United States, and the United Kingdom. Table 7.8 records the present numerical control limits in these jurisdictions.

In Ontario, the Regulation Respecting Asbestos, adopted in August 1982, set a control limit for a worker's time-weighted average exposure of 1 f/cc for chrysotile and all other forms of asbestos, except amosite which is subject to a 0.5 f/cc control limit, and crocidolite which is subject to a 0.2 f/cc control limit.[56] The TWA is to be calculated as the average exposure over a 40-hour week.[57] The short-term exposure limit, representing the maximum concentration to which a worker may be exposed for 15 minutes, is 5 times as great as the corresponding TWA control limit for each asbestos type.[58] In mixed asbestos fibre atmospheres, the control limit applied to all fibres is the one applicable to the most strictly regulated fibre type present.[59]

Most of the other Canadian provinces have tended to use the threshold limit values (TLVs) of the American Conference of Governmental Industrial Hygienists (ACGIH) as a basis for regulating asbestos. Thus, the present occupational standard in British Columbia, Alberta, New Brunswick, Nova Scotia, and Newfoundland is 2 f/cc for chrysotile, 0.5 f/cc for amosite and tremolite, and 0.2 f/cc for crocidolite.[60] Prince Edward Island seems not to have adopted a specific asbestos regulation. In Manitoba, a limit of 0.1 f/cc for all types of asbestos is apparently being considered.[61]

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[56] Regulation Respecting Asbestos, s. 4(1).

[57] See Schedule to Regulation Respecting Asbestos.

[58] Regulation Respecting Asbestos, s. 4(2).

[59] Ibid., s. 4(3).

[60] British Columbia — Industrial Health and Safety Regulations, B.C. Reg. 585/77, as am. by B.C. Reg. 374/79; Alberta — General Accident Prevention Regulation, Alta. Reg. 267/76, s. 79; New Brunswick — Occupational Safety Code Regulations (N.B. Reg.) 80-82, s. 8(4); Nova Scotia — Occupational Health Regulations R.&R., S.N.S. 76, 1510, s. 4(1); Newfoundland — Occupational Health and Safety Regulations, Nfld. Reg. 140/79, s. 31(6).

[61] Telephone communication between Mr. Dennis Nikkel, Industrial Hygiene Section, Workplace Safety and Health Division, Department of the Environment and Workplace Safety and Health, Government of Manitoba and Royal Commission on Asbestos Staff.

Table 7.8

Summary of Asbestos Control Limits, 1983
(All in f/cc. Membrane filter method used, except as noted.)

		Fibre Type		
Jurisdiction	**Duration of TWA**	**Chrysotile and Others**	**Amosite**	**Crocidolite**
Ontario	40	1	0.5	0.2
Quebec	Not specified	2 (all types) (also 0.2 mg/m^3, all types)		
Majority of other Canadian provinces	8	2	0.5 (and tremolite)	0.2
U.S.A. (OSHA)	8	2 (all types)*		
United Kingdom	4	1	0.5	0.2 (banned)
ACGIH	8	2	0.5 (and tremolite)	0.2

Note: *While this Commission's Report was in press, the U.S. Occupational Safety and Health Administration issued an Emergency Temporary Standard of 0.5 f/cc for all types of asbestos, which was subsequently stayed by court order. See U.S., Department of Labor, Occupational Safety and Health Administration, "Occupational Exposure to Asbestos; Emergency Temporary Standard," 29 CFR Part 1910, 48 FR 51086-51140, 4 November 1983.

Saskatchewan has banned the use of crocidolite in the workplace[62] and has a general requirement that hazardous dust be maintained below current ACGIH levels.[63] Saskatchewan attempts to achieve the best practicable means of control and claims to have enforced control measures even below levels of 0.02 f/cc, although it is not clear how air monitoring of such levels is achieved.[64]

In Quebec, there is a control limit set by regulation under *The Quebec Mining Act* for asbestos exposure to all types of asbestos of 2 f/cc longer than 5 microns.[65] However, it appears that this control limit is deemed to be met if a gravimetric measurement of asbestos dust in the air yields less than 0.2 milligrams per cubic metre of air.[66] In any event, the actual fibre count (as opposed to the mass measurement) may not exceed a short-term ceiling value of 5 f/cc in the air. The same limits apply to manufacturing.[67]

In the United States, the Occupational Safety and Health Administration adopted, in July of 1976, a control limit of 2 f/cc for all types of asbestos measured over an 8-hour TWA with a 10 f/cc ceiling level during one hour.[68] In 1976, the National Institute for Occupational Safety and Health recommended to OSHA an exposure limit of 0.1 f/cc, which has not been adopted. In the summer of 1983, OSHA was under pressure to reconsider and tighten the 2 f/cc control limit.

In 1979, the U.K. Advisory Committee on Asbestos recommended control limits of 0.2 f/cc over a 4-hour period for crocidolite, 0.5 f/cc for amosite, and 1.0 f/cc for chrysotile.[69] The Advisory Committee recommended that there should in addition be a requirement to reduce exposure to the minimum that is reasonably practicable.[70] In 1978, it had recommended a ban on the spraying of asbestos-containing insulation.[71] The Health and Safety Executive announced on August 27, 1982 that the Health and Safety Commission had adopted the recommended control limits, to take effect January 1, 1983. The Commission also decided to prohibit the spraying of asbestos, to prohibit the use of asbestos in insulation, and to prohibit the import, use, and marketing of crocidolite and products containing it.[72]

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[62] *The Occupational Health and Safety Act*, R.S.S. 1978, c. 0-1, s. 109, and Asbestos Regulations, Sask. Reg. 58/75, s. 2(2).

[63] Accident Prevention Regulations, Sask. Reg. 283/69, s. 16.02.

[64] RCA Transcript, Evidence of Mr. Robert Sass, 15 June 1982, Volume no. 40(A), p. 9.

[65] Regulation amending the Regulation respecting the safety of the workmen and sanitary conditions in mines and quarries, O.C. 2308-77 (1977) 109 *Gazette Officielle* II 3793; Q. Reg. 77-405.

[66] Ibid. One milligram is one million nanograms, so this standard equals 200,000 ng/m^3.

[67] Regulation respecting the quality of the work environment, O.C. 3845-80 (1981) 113 *Gazette Officielle* II 87.

[68] 29 CFR 1910. 1001.

[69] U.K., Advisory Committee on Asbestos, *Asbestos — Volume I: Final Report of the Advisory Committee*, Recommendations 14, 17, and 15, pp. 74, 78, 77 respectively.

[70] Ibid., Recommendation 10, p. 73.

[71] U.K., Advisory Committee on Asbestos, *Asbestos — Work on Thermal and Acoustic Insulation and Sprayed Coatings* (London: Her Majesty's Stationery Office, 1978); and U.K., Health and Safety Commission, *Consultative Document: Asbestos Insulation and Coating — Draft Regulations* (London: Her Majesty's Stationery Office, 1981), p. 3.

In Europe, the Council of the European Communities is currently considering asbestos control limits. Each member country would be expected to adopt control limits equal to or more strict than any directive finally made by the Council. No control limits have yet been adopted; however, the limits that have been discussed are an 8-hour TWA of 1.0 f/cc for all types of asbestos other than crocidolite, and a more strict limit for crocidolite.

E. Criteria for Setting Control Limits

What criteria should be used for determining the maximum asbestos fibre level to which workers may be exposed in Ontario? Labour organizations appearing before this Commission made various arguments that exposure to asbestos should be reduced as far as possible. The Labour Council of Metropolitan Toronto recommended that all "non-essential" uses of asbestos be phased out by June 1982, and that essential uses be defined as those for which there is no safe substitute available.[73] Where asbestos use continues, the exposure should be limited to the lowest amount measurable. In a written submission to this Commission, the United Steelworkers advocated reducing exposure to asbestos fibres to "an irreducible minimum."[74] The Ontario Federation of Labour (OFL) recommended that all non-essential uses of asbestos be phased out within one year, with all uses phased out as soon as safe substitutes are available, but no later than four years.[75] On the other hand, the OFL argued that substitutes should not be used unless proved safe, and that fibreglass has not been so proved.[76] Few substitutes are likely to pass this rigorous test in the near future. While these positions fall short of demanding an immediate end to all uses of asbestos in Ontario, they do explicitly reject the notion of "acceptable risk" and the use of cost-benefit analysis to determine control limits.

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[72] U.K., Health and Safety Executive, "New Safety Limits for Asbestos," *News Release*, 27 August 1982. In a copy of a telex to the British Embassy in Washington and dated 24 August 1983, sent by Mr. Cyril D. Burgess of the U.K. Health and Safety Executive to the Royal Commission on Asbestos, it was stated, *inter alia*, that the Health and Safety Commission had decided that: "1. Exposure limits for chrysotile to be reduced to 0.5 fibres per ml and for amosite to 0.2 fibres per ml effective from 1 August 1984. 2. A ban should be placed on the importation and use in manufacture of amosite and products containing it. Prohibition regulations should be brought forward for amosite and crocidolite to come into effect on 1 June 1984. . . ."

[73] Labour Council of Metropolitan Toronto, Written submission to the Royal Commission on Asbestos, #13, January 1981, p. 21.

[74] United Steelworkers of America, Written submission to the Royal Commission on Asbestos, #42, February 1981, p. 3.

[75] Ontario Federation of Labour, Written submission to the Royal Commission on Asbestos, #35, January 1981, p. 124.

[76] Ibid., p. 78.

A number of parties appearing before this Commission explicitly raised the issue of substitutes for asbestos, and we consider it clearly relevant to our terms of reference. Of course, it is quite impossible for us to assess the thousands of uses of asbestos and to form a judgement on whether a substitute with adequate performance characteristics is available at a reasonable price for each use. We have made such judgements in some cases, as when we endorse the ban on spraying asbestos-containing fireproofing in buildings (see Chapter 9); or when we recommend a ban on the sale of consumer products containing loose asbestos likely to release fibres during normal use or handling (see Chapter 11). In many cases, however, the evidence on the cost and performance of substitutes is mixed and does not lead to robust conclusions. Moreover, the evidence on the health effects of widely discussed substitutes for asbestos in manufacturing, which we review in Chapter 6, reveals few cases where an available substitute has been "proved safe," and provides no clear answer to the disturbing possibility that some substitutes may expose workers to fibres of dimensions similar to those we have found hazardous for asbestos. In Chapter 15 we discuss the responsibility of government to improve society's knowledge of the relative safety of asbestos substitutes through improved processes of hazard identification and assessment. For present regulatory purposes, we attach great importance to the fact that the hazardous nature of asbestos is known. We consider it better to regulate a known hazard rigorously than to compel the use of substitutes whose hazardous nature, being unknown, is not subject to regulation to a similar degree of rigour. Strict control limits can be set for asbestos exposure; current knowledge in the realm of hazard identification does not permit the setting of similarly strict limits for substitutes. If a firm can maintain worker exposure at the levels required by a rigorously enforced control limit, the alternative of forcing the firm to use a substitute is a leap into the unknown unless that substitute has been proved safe. If the control limit cannot be achieved, it is preferable to prohibit the firm from operating than to force it to use a substitute that has not been proved safe. Such prohibition is a powerful incentive to a firm to find a substitute that can indeed be proved safe and in our view is more effective than a blanket requirement that all firms using asbestos should search for asbestos substitutes.

We have reviewed a substantial body of literature and the reports prepared by researchers for this Commission, looking for guidance on an appropriate basis for setting occupational health standards. While this literature has offered a variety of ways of analyzing the problem, it has not provided a direct means of reaching quantitative conclusions. In their study for this Commission, Professor Carolyn J. Tuohy and Professor Michael J.

Trebilcock applied four frameworks for analyzing alternative policies for workplace control of asbestos: a scientific framework, an economic framework, a political framework, and an ethical framework.[77] They concluded that each of these frameworks can contribute to an evaluation of alternative policies, although they will frequently lead to results inconsistent with each other. Professor Tuohy and Professor Trebilcock emphasized that the process by which standards are set is important, and that the parties most directly affected by a standard should be centrally involved in designing that standard itself. They suggested that the standard-setting process inevitably involves a balancing of costs and benefits, whether this is done explicitly or implicitly. They were unable, however, to provide a formula that would define the "correct" standard for occupational exposure. In their study for this Commission, Professor G. Bruce Doern, Professor Michael Prince, and Mr. Garth McNaughton discussed the processes used for setting occupational and environmental standards.[78] They, too, derived conclusions about the appropriate process for standard-setting, but did not attempt to suggest the formula by which a standard can be selected. Other volumes have been written on risks, risk assessment, and decisions regarding risk. William D. Rowe developed an elaborate analytical framework for evaluating risks and assessing society's attitude towards those risks in *An Anatomy of Risk*.[79] The book is very informative, but the chapter called "Determination of Acceptable Levels of Societal Risks" contains some methodology which includes important value judgements that are either made by the author or left for the analyst to supply. While recourse to such a framework may lead to some understanding of the relative risks that exist in different situations, we do not see in this methodology any shortcut for arriving at an appropriate level of risk to be allowed in workplace situations. A book edited by Richard C. Schwing and Walter A. Alberts, Jr., called *Societal Risk Assessment*, contains many papers on risk assessment, the acceptability of risk, and many methods for calculating risk levels in different situations.[80] Cost-benefit analysis and decision analysis are discussed at length. Risks arising in various situations are presented and compared. Once again, however, we do not see here a single methodology that would lead us to a "correct" risk level or control limit for occupational exposure to asbestos.

Our conclusion is that setting a control limit requires first an assessment of the risk associated with different control limits. Once this risk assessment has been completed, a variety of factors must be considered in selecting an appropriate control limit. We cannot escape making difficult judgements about risks that may be allowed in the workplace, nor can we conceal such judgements by the use of elaborate mathematical calculations.

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[77] Carolyn J. Tuohy and Michael J. Trebilcock, *Policy Options in the Regulation of Asbestos-Related Health Hazards*, Royal Commission on Asbestos Study Series, no. 3 (Toronto: Royal Commission on Asbestos, 1982).

[78] G. Bruce Doern, Michael Prince, and Garth McNaughton, *Living with Contradictions: Health and Safety Regulation and Implementation in Ontario*, Royal Commission on Asbestos Study Series, no. 5 (Toronto: Royal Commission on Asbestos, 1982).

[79] William D. Rowe, *An Anatomy of Risk* (New York: John Wiley & Sons, 1979), chap. 18.

[80] Richard C. Schwing and Walter A. Alberts, Jr., eds., *Societal Risk Assessment* (New York: Plenum Press, 1980).

There exist several approaches that could be applied to determining the appropriate control of worker exposure to asbestos. Exposures could be reduced: (i) until all health risks are eliminated; (ii) to the measurement limit, the level below which current measurement technology does not allow enforcement of standards; (iii) to the technological limit, the point below which current technology for controlling exposure does not permit further reductions; (iv) until the cost of further reductions exceeds the health benefits therefrom; or (v) until the remaining health risks are either not significant, or are acceptable. We will consider each of these approaches as a basis for controlling worker exposure to asbestos.

Eliminating All Risks — Desirable though it may be in principle, the idea of totally eliminating risk is not attainable in the real world. Since virtually every activity, in the workplace or the environment, involves some risk, it will always be necessary to approach any matter of health and safety in terms of relative risk, rather than in terms of absolutely eliminating risk. We conclude, based on the medical evidence discussed in Chapters 4 and 5, that the best conservative estimate of the dose-response relationship between asbestos exposure and lung cancer and mesothelioma is a straight line through the origin. This means that excess deaths due to cancer are not eliminated so long as exposure to asbestos fibres is not eliminated. At very low fibre levels, the health effect may be very small. Indeed, it may approach zero, without ever attaining it. Absolute freedom from risk is not attained in other aspects of occupational health and safety. Accordingly, we do not believe that absolute freedom from risk is an appropriate criterion for asbestos fibre control. The appropriate criterion must include a weighing of relative risks, discussed below.

Measurement Limit — We reject the concept of setting a control limit based upon measurement capability alone. In the first place, the definition of the measurement limit for a particular substance is an uncertain business in itself, with serious debate currently underway as to the lower limit of measurement capability. (See Chapter 7, Section B.) Even if agreement could be reached on the control limit that currently represents the lowest level that could reliably be measured, tying the control limit to measurement technology leaves workers and industry at the whim of uncertain technological change. Advances might not be made for a decade, or measurement capability might improve a hundredfold within a few years. The health consequences of a given exposure are not affected by measurement technology, nor are the costs of control. However, if it is concluded that the appropriate level of exposure is well below current measurement capabilities, then perhaps the substance should not be used at all. To tie the control limit solely to measurement capability is to abrogate the responsibility for making a difficult decision; namely, to prohibit use of a substance that poses an unacceptable risk even when exposures are below the level of measurement capability.

Best Technology — For similar reasons, we reject the explicit setting of a control limit based solely upon the best available technology. The problem with this approach is that it leaves protection of the worker to the uncertain outcome of research and development in the technology of worker protection. We do not believe that responsible governments should abandon their decision-making responsibilities to the uncertain outcomes of engineering research and development. If the appropriate control limit cannot currently be achieved, then perhaps the substance should not be used at all. If, on the other hand, a satisfactory control limit does not require use of the most sophisticated available technology, unnecessary expense should be avoided.

Furthermore, a careful examination of the application of control limits which purport to require that the best technology be installed will reveal that in fact cost considerations are often taken into account. There are few occupational or environmental problems where further exposure reduction is not technologically achievable at some — perhaps astronomical — cost, in worker inconvenience, in money, or both. If one combines complete process enclosure with worker isolation and elaborate respiratory protection for maintenance workers, worker exposure levels to asbestos fibres could conceivably be reduced nearly to zero. We believe, however, that if a "best available technology" control limit were adopted, its implementation would not lead to near zero exposure, but rather to an exposure that was limited by the cost of more extensive controls. We think that the adoption of a best available technology approach would be deceptive because we believe that cost considerations would manage to insert themselves in the implementation of such a control limit.

Cost-Benefit Analysis — We do not accept the use of cost-benefit analysis as the sole means for identifying a specific control level. Submissions to this Commission have identified the severe limitations of cost-benefit analysis for yielding a single, unambiguous number representing the appropriate control level. Our discussion of control costs in Chapter 6 suggests substantial uncertainty about actual cost levels, and the discussion of health effects in Chapters 4 and 5, and in Section C of this chapter, suggests an enormous range of uncertainty in the possible health outcomes from various exposure levels. While a weighing of cost and benefit information may be useful in deliberations about the selection of an appropriate control level, it cannot identify a unique control level. Nor should cost-benefit analysis alone determine the control level even if enormous uncertainty did not exist, since factors which cannot be entered in a quantitative form in a cost-benefit analysis should play an important role in reaching occupational health and safety judgements.

We believe that the concept of relative risk with the addition of some of the approaches just discussed constitutes a basis for controlling worker exposure to asbestos to acceptable levels. The concept of relative risk suggests that the significance of a risk can be determined by comparison with other risks that are faced by workers. If a risk is sufficiently small, compared to other risks faced by workers, it might be regarded as insignificant and hence not warranting further action to reduce the risk. We will conclude in the next section that the risks from asbestos-related disease faced by Ontario workers under the recently adopted asbestos control limits are significant; this requires in turn an assessment of whether these significant risks are acceptable.

One witness who appeared before this Commission suggested an absolute, rather than a relative, approach to determining the significance of a risk. Dr. Bailus Walker, Jr., after acknowledging the difficulty of determining when a risk becomes significant, suggested in an article he had written that a risk higher than 1 in 100 should be banned, a risk lower than 1 in 100,000 should be regarded as trivial, and a risk between these two levels should be the subject of public education warnings and efforts at reduction.[81] The implication is that a risk of cancer death of 1 in 100,000 is an insignificant risk, while a risk of 1 in 100 is significant, in fact so significant that it is unacceptable. Dr. Walker was not clear, however, on where between these widely separated points the insignificant becomes significant, nor on whether a risk of 1 in 100 refers to the probability of death in one year or in a lifetime. We are not convinced that such measures of absolute risk are helpful in formulating occupational health policy.

The acceptability of a risk depends upon a social value judgement made by the affected parties or by the legislature or by society as a whole. Numerous significant risks have been judged acceptable by the individuals who face them, the government agencies that regulate them, and society as a whole. Thus, while the risk of fatal accidents in mining is significant, that risk has been found acceptable in that the mining industry continues to operate; it has not been shut down by government nor by its own workers because of the risks faced by miners. To be sure, the acceptance of this risk is conditional. It is conditional on the continuation of efforts to reduce the risk. This is one reason for the frequency and thoroughness of investigations into mine safety.[82]

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[81] Bailus Walker, Jr., "Occupational Cancer," *Journal of Environmental Health* 44:4 (January/February 1982): 179.

In assessing the acceptability of a risk, we will compare the risks caused by an asbestos exposure to the risks faced by workers from other sources, principally the risk of accidents. When a disease risk is below the accident risk, then it may (but need not) be considered acceptable. When the disease risk exceeds the accident risk, then it approaches the range of being not acceptable. Within this range, other factors may also be considered in assessing acceptability.

In defining significant and acceptable risk, we have referred to a comparison with other workplace risks. Would it be appropriate to consider non-workplace risks as well? Many studies have compared the risk of fatal injury in a wide variety of activities, and have compared the cost of additional life saving in those activities. Such studies show that a number of sporting activities are far more hazardous than most work activities. These facts are used to argue that individuals willingly face considerable risks, and that it is extravagant to be so protective in the workplace. The counterargument is that there is a difference between voluntary assumption of risk in personal activities and the exposure to risk in a job which, once career and place-of-employment decisions have been made, is largely involuntary. We believe that it is not productive to compare risks in widely divergent types of activities because of the difficulty of interpreting the factors that influence those different choices. Furthermore, the risks that are allowed in different activities vary enormously. We are reluctant to spend small amounts to reduce the highway death toll further, yet we spend large amounts of money to avoid occupational health risks. For example, John D. Graham and James W. Vaupel found that in the United States the median cost per life saved in various programmes was $64,000 in motor vehicles and highways; $102,000 in medical programmes; $50,000 in consumer protection; $2.6 million in environmental protection; and $12.1 million in occupational health and safety programmes.[83] Not all the programmes analyzed have in fact been adopted by the U.S. government, but these figures indicate the range of costs associated with saving a life in various programmes in the United States. Thus, a comparison of the risks faced by a variety of groups in a variety of activities provides little guidance as to whether workplace risks are significant or acceptable. We will, therefore, compare asbestos-related risks only with other workplace risks.

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[82] See Ontario, *Report of the Royal Commission on the Health and Safety of Workers in Mines* (Ham Report), James M. Ham, Commissioner (Toronto: Ministry of the Attorney General, 1976), and Canada/Ontario, *The Report of the Joint Federal-Provincial Inquiry Commission into Safety in Mines and Mining Plants in Ontario: Towards Safe Production* (Burkett Report), Kevin M. Burkett, Chairman, 2 vols. (Toronto: April 1981).

[83] John D. Graham and James W. Vaupel, "Value of a Life: What Difference Does it Make?" *Risk Analysis* 1:1 (March 1981): 91.

While we believe that some risks that are significant may be acceptable, we believe that some significant risks may be so substantial that they are unacceptable. It goes without saying that the illness and death that have been caused by high asbestos exposures in the past in Ontario and elsewhere in the world must be judged completely unacceptable. Well below this, the level of significant risk that is unacceptable depends in part upon the type of risk and the perception of the risk. Jobs involving risks to safety that are obvious to the worker, and which the worker can evaluate and judge intelligently, may be allowed to continue at a higher level of risk than jobs in which the risk is not obvious. In the former case, it can at least be argued that the worker can make an informed choice about accepting the risks with the job and can take action to reduce those risks when on the job. Thus, a higher risk of fatal accidents has been tolerated in mining and construction than in manufacturing, in part because the accident risks in mining and construction are well known to the workers. Workers who enter manufacturing do not, in general, expect nor accept such high accident risks. More important, the risk of disease is neither visible nor obvious to workers. The risk of long-latency diseases such as asbestos-related cancer, when caused by dust levels that are hardly visible to the naked eye, as are current asbestos exposures in manufacturing, would be completely unknown to a worker unless he were specifically informed of the risk. If the worker cannot be expected to know about the risk or form an intelligent opinion about the severity or magnitude of the risk, then the socially acceptable risk level must be considerably lower than if the risk is obvious.

When the risk of disease is significant, the determination of the acceptable risk level must depend upon whether the risk is known to, and understood by, the worker, the health effects of alternative exposure levels, the technology available for controlling that exposure, and the cost of control. If real reductions in the health risk can be achieved at a reasonable cost, then those reductions should be made mandatory by lowering the control limit.

If the risk of disease becomes insignificant, there is little gain from further control. If the cost of control is already high, then imposing still more strict control limits might not only cause costly short-term dislocations for both labour and industry, but might alternatively divert resources from attacking other health and safety problems in the plant where the improvement in worker health might be greater.

F. Recommendations on Workers' Exposure

How do the Ministry of Labour's recently adopted control limits fare under our criteria? Table 7.9 repeats the estimated risk of mortality from cancer predicted by the studies presented in Table 7.6 in Section C above for short-term, normal exposure (10 years' duration at one-half the control limit) and for long-term, high exposure (25 years' duration at the control limit). We may compare these estimated asbestos-related mortality rates to other risks faced by workers to determine whether the risk is significant. We will consider each process separately. It must be remembered that considerable uncertainty is associated with all of the estimated risks, so each number discussed should be regarded as representing a range of possible risks.

How do these risks compare with other occupational risks? *The Report of the Joint Federal-Provincial Inquiry Commission into Safety in Mines and Mining Plants in Ontario* showed accident fatality rates per million person-hours of work equal to 0.038 in manufacturing, 0.160 in construction, and 0.516 in mining.[84] If a workforce of 1,000 workers were employed for 10 years, working 2,000 hours per year, they would work a total of 20 million person-hours. Twenty-five years of work would yield 50 million person-hours. The number of hours of work may be multiplied by the accident fatality rate to determine the approximate fatal accident risk for such a workforce. Twenty million person-hours of work in manufacturing would result in an average accident risk of about 0.8 fatalities for 1,000 workers employed for 10 years. The same amount of work in construction would result in an average fatal accident risk of 3.2, and in mining, a risk of 10.3. The corresponding risks for 50 million hours of work would be 1.9, 8.0, and 25.8. These accident risks are presented at the bottom of Table 7.9. We do not have data on the person-years of life lost for these sectors, but accident deaths tend to occur at an early age, while asbestos deaths occur at a later age because of latency. Thus, perhaps 4 times as many person-years of life might be lost in accident deaths as in the same number of asbestos-related cancer deaths. While this is a statistical fact, we will rely primarily on the raw mortality data, rather than losses in person-years of life in making our risk comparisons.

We stated in Section E above that a risk was insignificant only if it was far below the level of other risks faced by workers generally. Under the 1982 Ontario control limits, the lowest risk for any process or fibre type is 0.06 possible deaths per 1,000 workers exposed for 10 years, while the manufacturing accident rate is approximately 0.8. (See Table 7.9.) We believe that this difference is not so great as to render the mortality risk from asbestos exposure insignificant. We will therefore consider, for each process, whether this significant disease risk is acceptable.

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[84] Canada/Ontario, *The Report of the Joint Federal-Provincial Inquiry Commission into Safety in Mines and Mining Plants in Ontario: Towards Safe Production*, vol. 2: *Statistics and Research Reports*, p. 15.

Table 7.9

Risk Assessment by Process and Fibre Type Recent Ontario Control Limits

Process	Fibre Type	Study Used	Applicable Control Limit (f/cc)	Mortality Risk per 1,000 Workers at the Control Limit	
				Short-term, Normal Exposure*	Long-term, High Exposure**
Mining	Chrysotile	J.C. McDonald	1.0	0.1-0.2	0.4-0.9
Manufacturing a) General (friction products)	Chrysotile	Berry	1.0	0.2	1.1
b) Textiles		Dement	1.0	16	76
		Peto***	1.0	6.2	26
Manufacturing	Mixed (crocidolite)	Enterline	0.2	0.06	0.3
		Peto***	0.2	1.3	5.3
		Finkelstein	0.2	17	57
Insulation work	Mixed (amosite)	Selikoff	0.5	4.1	16
Workplace accident risks				**10 Years**	**25 Years**
Manufacturing accidents (0.038 x 20, 50)				0.8	1.9
Construction accidents (0.16 x 20, 50)				3.2	8.0
Mining Accidents (0.516 x 20, 50)				10.3	25.8

Notes: *Assuming a 10-year average duration of exposure at one-half the control limit.

**Assuming a 25-year duration of exposure at the control limit.

***There is uncertainty whether this textile plant used crocidolite as well as chrysotile.

SOURCE: Asbestos risks derived from Table 7.6. Accident risks derived from Canada/Ontario, *The Report of the Joint Federal-Provincial Inquiry Commission into Safety in Mines and Mining Plants in Ontario: Towards Safe Production* (Burkett Report), Kevin M. Burkett, Chairman, vol. 2: *Statistics and Research Reports* (Toronto: April 1981), p. 15

F.1 Mining

In asbestos mining, we estimate that the short-term, normally exposed worker faces an asbestos-related mortality risk of 0.1 to 0.2 per 1,000 workers, while the long-term, highly exposed worker faces a mortality risk of 0.4 to 0.9 per 1,000 workers. Even the highly exposed worker's risk is only 4% of the risk of accidental death in mining, while the normally exposed worker's risk is 1 to 2%. The mining worker's asbestos-related mortality risk is even below the accidental mortality risk in manufacturing.

Which comparison is relevant for determining the acceptability of this disease risk? While miners clearly accept higher risks of accidental death than do workers in manufacturing, we see no reason to believe that this acceptance extends to non-obvious and long-delayed disease risks. Although the risk of asbestos-related disease mortality in mining is small compared to the risk of accidental death in mining, it is not small when compared to the risk of accidental death in manufacturing. We are uneasy about adopting the high mining accident rate as a standard of comparison for evaluating health risks. We choose the risk of accidents in manufacturing as the relevant basis of comparison for determining the acceptability of health risks in mining.

Is this significant health risk acceptable? We find that this health risk falls well within the range of occupational health risks that have been found acceptable by society. This risk is a small proportion of the risk of accidental death in mining, and is below the risk of accidental death in manufacturing. (See Table 7.9.) We, therefore, endorse the application of the 1 f/cc chrysotile control limit to the mining industry, as is presently provided in the Regulation Respecting Asbestos.[85] Our finding of acceptability is, however, contingent upon workers being aware of the nature and magnitude of the risk of asbestos disease posed by their exposure. No worker should be exposed to a significant risk without his knowledge.

F.2 Chrysotile Manufacturing

(a) General

With respect to general chrysotile manufacturing processes, where the chrysotile is quickly encapsulated, as exemplified by friction products manufacturing, we estimate that the mortality risk faced by the short-term, normally exposed worker is 0.2 deaths per 1,000 workers, while the risk to the long-term, highly exposed worker is about 1.1 per 1,000 exposed workers. This may be compared to risks from accidental deaths in manufacturing of 0.8 to 1.9 deaths per 1,000 workers, although the asbestos-related deaths occur at a far older age on average than those from accidents.

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[85] Regulation Respecting Asbestos, s. 4(1). See also, Chapter 3, note 85 for amplification.

Is this significant risk acceptable? We find that this health risk falls well within the range of occupational health risks that have been found acceptable by society. The risk is on average less than that from accidental death in manufacturing, and the number of life-years lost is substantially less. The 1,200 or so Ontario manufacturing workers now exposed to asbestos could work for 10 years and there would be only a one in four chance of a single premature death among the 1,200 from this exposure. The 1 f/cc chrysotile control limit reduced the allowable exposure of workers in Ontario by 50% compared with the guideline which previously existed and to 10% or less of the exposures experienced during the 1950s. We have seen no evidence of high disease rates from chrysotile asbestos manufacturing in Ontario in the past, and the recently adopted control limit will greatly reduce such disease risks as existed. Our finding of acceptability is, however, contingent upon certain conditions being met:

(i) if compliance with the control limit is such that the exposure of the average worker is significantly less than the control limit;

(ii) if workers are aware of the nature and magnitude of the health risks posed by this exposure — no worker should be exposed to a significant risk without his knowledge; and

(iii) if the Regulation Respecting Asbestos is enforced so that plants which can achieve exposure levels well below the control limit will do so.

We observe that the Regulation Respecting Asbestos made under the *Occupational Health and Safety Act* in August of 1982, in section 4(1), not only states numerical exposure limits for the three major types of asbestos, but precedes the statement of these limits with the requirement ". . . that the time-weighted average exposure of a worker to airborne asbestos is reduced to the lowest practical level and in any case shall not exceed. . . ." We interpret "the lowest practical level" to be a requirement that employers reduce exposures below the numerical control limit wherever this is technologically and economically reasonable. The implementation of "the lowest practical level" is discussed further in Chapter 8 of this Report.

(b) Textile Spinning and Weaving

Next we consider manufacturing of chrysotile that is not encapsulated, represented specifically by the textile spinning and weaving industry. The estimated mortality risks indicated in Table 7.9 are 6.2 to 16 per 1,000 workers for the short-term, normal exposure, and 26 to 76 per 1,000 workers for the long-term, highly exposed worker under the recently adopted Ontario control limit. Such risks are clearly substantial, when compared to the accidental death risk in manufacturing of 0.8 to 1.9 per 1,000 workers. These risks are even greater than the risk of death from accidents in mining. We find that this health risk falls beyond the range of occupational health risks that have been found acceptable by society. The normally exposed worker would be exposed to cancer risks more than 10 times greater than the risk of accidental death, and the highly exposed worker would face risks that might be 25 times greater than the accidental death risk. We do not believe that workers in Ontario should be asked to accept such substantial risks. We are not convinced that an information programme for workers could effectively communicate to them the magnitude of the long-term health risks they face, so that they could knowingly accept the risk.

At the present time, there are no substantial textile manufacturing operations in the province of Ontario. There is some weaving of asbestos packings from yarn spun outside the province, but here the yarn is generally coated with liquids before being woven. There is also some manufacture of textile products from cloth woven outside the province. However, there is the possibility that at some time in the future a textile spinning and weaving plant might be constructed in Ontario to operate under the 1 f/cc control limit. Having concluded that the risks involved in such textile operations are not acceptable, we believe that the Ministry of Labour should be in a position to prohibit the operation of such a plant unless it was assured that the health risks faced by workers were acceptable. Such assurance might arise if the risks were lowered by a factor of 25, so that they approached the same range as those of other chrysotile manufacturing. This might be achieved by a control limit for textile plants of 0.04 f/cc or by a control limit of 0.1 f/cc combined with a commitment to employ only non-smoking workers. However, at the present time, the standard workplace measurement method would be inadequate to enforce exposure levels below 0.1 f/cc. Alternatively, such assurance might arise if subsequent studies of chrysotile textile plants were to demonstrate that the Dement study has seriously overestimated the risks and that the true risk is considerably less. In light of these considerations, we recommend that:

7.11 The Government of Ontario should adopt regulations that would prohibit the operation of asbestos textile spinning and weaving plants in Ontario without the approval of the Ministry of Labour. This approval should be granted only if the Ministry is satisfied that the health risks faced by workers in the plant are far lower than the unacceptable risks estimated in this Report.

(c) Asbestos-Cement Production

When we come to asbestos manufacturing involving mixed exposures, we shall recommend that the use of crocidolite and amosite be prohibited and thus in effect ban the manufacture of asbestos-cement pipe when chrysotile is mixed with either amosite or crocidolite. There remains the question of whether asbestos-cement manufacturing using chrysotile only should be regulated in the same manner as general chrysotile manufacturing or should be treated as a special case. The tragic health experience at the Scarborough Johns-Manville plant was one that involved exposure to crocidolite. Can it be, however, that this experience can also be attributed to peculiarities associated with the pipe manufacturing process, independent of the type of fibre used? The studies of asbestos-cement manufacturing without crocidolite suggest that the answer to this question is negative. The Weill study of New Orleans asbestos-cement plants discovered no excess risk of respiratory malignancy among those workers not exposed to crocidolite, while workers with crocidolite exposure suffered elevated health risks.[86] The Thomas study of Cardiff asbestos-cement manufacturing workers not exposed to crocidolite showed low excess risks of respiratory malignancy.[87] (See Chapter 5, Section B.) The Enterline study of Manville workers revealed a low excess risk of lung cancer for the entire cohort, which is the basis for the Enterline data in Table 7.9 and in Table 7.3.[88] Notably, however, the portion of the cohort exposed only to chrysotile in asbestos-cement operations experienced a lung cancer risk lower than that of the cohort as a whole, while those exposed to crocidolite in asbestos-cement production experienced excess lung cancer risks twice those of the cohort as a whole.[89] While the Appendix to Chapter 7 does not separately analyze the groups of workers exposed to different fibre types in the Enterline cohort, we can interpret its analysis of the cohort as a whole as an overestimate of the risk faced by the chrysotile-only asbestos-cement workers. Table 7.3 shows mortality risks for the Enterline data of 0.3 for the short-term, normally exposed worker under a 1.0 f/cc control limit, and of 1.4 for the long-term, highly exposed worker. These risks are close to the risks in Table 7.9 for friction products, which we determined fall within the range of societal acceptability. We find on the available evidence that the health risks from asbestos-cement manufacturing with chrysotile only are roughly similar in magnitude to those from friction products manufacturing. We, therefore, endorse treating asbestos-cement product manufacturing with chrysotile only as general chrysotile manufacturing and thus subject to the 1 f/cc control limit.

What about chrysotile-using manufacturers other than friction product, asbestos-cement, and textile spinning and weaving plants? We endorse regulation by fibre type and process because we believe that these are reasonable proxies for those factors that influence the toxicity of respirable asbestos fibres: their size and perhaps surface chemistry. Because medical science does not now tell us precisely what it is that makes some exposures to asbestos more hazardous than others, we must rely on the few studies that have differentiated risks. Reviewing the list of other manufacturing operations in Ontario, we find mostly operations in which raw asbestos is mixed with other materials, usually liquids, and then worked on in some fashion. (See Chapter 6.) These operations seem more like friction products manufacturing than like textile spinning and weaving from the viewpoint of what is done with the fibres. In the absence of evidence to the contrary, we will presume that the health effects of these general chrysotile manufacturing operations are reasonably well represented by the risk assessments based on friction products, rather than by assessments based on textile spinning and weaving. We are somewhat reassured in this presumption by the fact that the claims data of the Workers' Compensation Board do not reveal alarming claims records from individual chrysotile manufacturing firms in Ontario. We, therefore, endorse the application of the 1982 Ontario control limit of 1 f/cc for chrysotile to all chrysotile manufacturing operations except textile spinning and weaving.

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[86] Hans Weill, Janet Hughes, and Carmel Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," *American Review of Respiratory Disease* 120:2 (August 1979): 345-354.

[87] H.F. Thomas et al., "Further Follow-up Study of Workers from an Asbestos Cement Factory," *British Journal of Industrial Medicine* 39 (1982): 273-276.

[88] Henderson and Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality."

[89] Ibid., p. 122.

F.3 Manufacturing — Mixed Exposures

(a) Crocidolite

We now consider manufacturing of asbestos products using asbestos fibres other than chrysotile. The estimated health risk faced by the short-term, normally exposed worker, at the recently adopted Ontario control limit of 0.2 f/cc for crocidolite, as shown in Table 7.9, ranges from well below the accidental death rate in manufacturing, to the 17 deaths per 1,000 workers predicted from the Finkelstein study of workers exposed to chrysotile and crocidolite in asbestos-cement pipe manufacturing in Scarborough. Under the 0.2 f/cc control limit, the long-term, highly exposed worker would face risks ranging from 0.3 to 57 per 1,000 workers.

Are these significant risks acceptable? The risks projected by Enterline are acceptable, given our conclusion regarding chrysotile manufacturing above, but the Enterline study was heavily weighted by workers exposed to chrysotile only. The crocidolite-exposed workers experienced excess lung cancer risks more than double those reflected in the data in Table 7.9. (See Chapter 5, Section B.) The risks projected by Finkelstein are clearly unacceptable. The risks projected by Peto are on the borderline of acceptability. We do not have to decide if the Peto risks are acceptable because our recommendation regarding textile plants, which considered the Dement study as well, would require lower control limits than dictated by the Peto study alone.

Another assessment of the risks of exposure to crocidolite in manufacturing may be made by comparing the disease experience of the asbestos workers at the Johns-Manville asbestos-cement pipe plant with that of workers in other environments thought to cause risks of workplace disease, and particularly cancers. Dr. Murray M. Finkelstein of the Ministry of Labour has studied the Johns-Manville workers and has compared their disease rates with those of workers at Falconbridge and Inco.[90] Table 7.10 shows the mortality of workers in five situations from all malignancies and from lung cancer. The Inco sinter plant workers experienced an excess mortality from all malignancies of 81%, while the asbestos-exposed Johns-Manville workers experienced an excess mortality of 200%. Considering lung cancer alone, the Inco sinter plant workers experienced an excess mortality of 250%, while the Johns-Manville asbestos-exposed workers had an excess risk of lung cancer of 380%. Thus, the experience of Johns-Manville pipe plant workers appears worse than that of workers where other major occupational disease problems have been found in this province.

The tragic health experience of those who worked in the Johns-Manville pipe plant is not unusual for workplaces where crocidolite is used. Other studies, discussed in Chapter 5, have shown that high disease rates have been recorded whenever the health of workers exposed to crocidolite has been studied. The Weill study of New Orleans asbestos-cement plants revealed no excess risk of respiratory malignancy among those workers not exposed to crocidolite, while those workers exposed to crocidolite faced significantly higher disease rates.[91] (See Chapter 5.) These high disease rates may result partly from the high dust concentrations raised by working with crocidolite, but they also occur because, as we conclude in Chapter 5, at comparable airborne fibre concentrations measured by optical methods, the hazard presented by crocidolite is greater than that presented by chrysotile. Since we do not have formal dose-response equations for the other crocidolite studies, we will rely on the Finkelstein study, which is based on local Ontario conditions, to reflect the general risk of working with crocidolite. If this introduces any error, it is to overestimate the risk of crocidolite exposure.

The Finkelstein data show that extremely low exposures would be required to reduce the health risks of working with crocidolite to a level comparable to the risks from working with chrysotile. For example, a control limit of 0.02 f/cc for crocidolite, 1/10 the current control limit, would yield risks of 1.7 for the short-term, normally exposed worker, and 5.7 for the long-term, highly exposed worker. This implies that at a given exposure level crocidolite may be more than 300 times as hazardous as chrysotile in

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[90] Murray M. Finkelstein, "Comparison of the Mortality Experience Among Employees of Inco and Falconbridge and Johns-Manville," Toronto, October 1982. (Mimeographed.)

[91] Weill, Hughes, and Waggenspack, "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing."

Table 7.10

Cancer Mortality in Selected Ontario Workplaces

Workplace	Excess Risk (%) All Malignancies	Lung Cancer
INCO		
Non-sinter	-2%	5%
Sinter	81%	250%
Falconbridge	1%	47%
Johns-Manville		
Non-asbestos	33%	0
Asbestos-exposed	200%	380%

SOURCE: Summary by Dr. Murray M. Finkelstein of the results of his own studies of Johns-Manville's Scarborough asbestos-cement workers and of the JOHC-INCO Mortality Study and the Falconbridge Mortality Study, both performed in 1982 at McMaster University. Presented in a letter from Dr. Ann E. Robinson, Assistant Deputy Minister, Occupational Health and Safety Division, Ontario Ministry of Labour to the Royal Commission on Asbestos, 1 October 1982.

brake manufacturing.[92] It is not possible at present either to achieve such low fibre levels in general manufacturing or to measure them with conventional methods, nor has it been suggested to this Commission by any witness that such low levels could be achieved or measured in the foreseeable future. It is thus necessary to prohibit the use of crocidolite in Ontario lest workers be exposed to unacceptable risks. We therefore recommend that:

7.12 The Government of Ontario should adopt regulations that would prohibit the use of crocidolite asbestos in Ontario workplaces without the approval of the Ministry of Labour. This approval should be granted only if the Ministry is satisfied that the health risks faced by workers exposed to this asbestos are far lower than the unacceptable risks estimated in this Report.

Can we use the risk projection based on asbestos-cement pipe manufacture with crocidolite to estimate the risk presented by crocidolite exposure in other manufacturing situations or in non-manufacturing situations? Because we believe that the process generating the absestos dust may affect the toxicity of the dust, it would be preferable to project risks in each process from epidemiological studies of that process. We do not have studies for other manufacturing processes involving crocidolite that are adequate for determining a dose-response function. In the absence of other information, we must assume that exposure to all crocidolite manufacturing processes presents the same health risks.

Even if no crocidolite is to be used in Ontario industry, some Ontario workers will continue to be exposed to crocidolite in some workplaces where crocidolite has already been installed, and must be disturbed, such as in buildings. Those workers who will be exposed to crocidolite in this way will in general be protected by the Proposed Regulation Respecting Asbestos on Construction Projects, which specifies control by procedure.[93] The use of respirators is often required, and the type of respirator depends upon the dust level in the workplace and the control limit that must be achieved for the worker. In the absence of a study that establishes a dose-response function for exposure to crocidolite in building insulation, we will rely again on the Finkelstein data. We believe that the respirators used should be sufficient to maintain the time-weighted average of a worker's breathing exposure at or below 0.02 f/cc, 1/10 of the current control limit. We therefore recommend that:

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[92] Compare Berry in Table 7.3, presenting a risk of 0.1 from an exposure of 0.2 f/cc to Finkelstein in Table 7.3, presenting a risk of 34 from an exposure of 0.2. The mortality rate *per f/cc* is over 300 times greater in the latter than in the former.

[93] Ontario, Ministry of Labour, Occupational Health and Safety Division, "Proposed Regulation Respecting Asbestos on Construction Projects and Related Codes," presented at a Public Meeting, 17 January 1983, Toronto. (Mimeographed.) This "Proposed Regulation . . ." is a revision of the "Notice of Proposed Regulation: Designated Substance — Asbestos on Construction Projects," *The Ontario Gazette*, vol. 115-33, Saturday, 14 August 1982, pp. 3194-3197. See Chapter 10 for recommendations regarding this proposed regulation.

7.13 The Code for Respiratory Equipment for Asbestos on Construction Projects should provide for the use of respiratory protection that will ensure that the worker's exposure does not exceed 0.02 fibres per cubic centimetre where crocidolite asbestos is in the air.

This does not require that fibre levels of 0.02 f/cc be measured. Instead, the actual workplace fibre concentration which may exceed 1 f/cc must be measured, and a respirator chosen with a factor of protection that would expose the worker to no more than 0.02 f/cc.

(b) Amosite

In Chapter 5 we conclude that the health risk from exposure to a given measured fibre concentration of amosite was more severe than that from exposure to a similar concentration of chrysotile, but probably somewhat less severe than that presented by exposure to the same measured concentration of crocidolite. Unfortunately, we have little direct evidence of the health effects of amosite from quantitative epidemiological studies. The Enterline study of Johns-Manville factory workers included some workers exposed to amosite as well as to chrysotile, and some workers exposed to crocidolite. As we report in Chapter 5, Enterline found that exposure to amosite alone created greater health risks than exposure to a similar concentration of chrysotile alone, but he attached little significance to this result because he also found that exposure to a mixture of amosite and chrysotile was no more hazardous than exposure to chrysotile alone. His data do not, therefore, form a sound basis for projecting health risks arising from amosite exposure in manufacturing.

The major analysis of the health effects of amosite is found in the Selikoff studies of insulation workers who installed and removed asbestos-containing insulation in the United States and Canada. These workers were exposed to chrysotile and to a substantial amount of amosite in their work. While it is entirely possible that manufacturing processes using amosite might create risks different from insulation work involving amosite, in the absence of appropriate manufacturing data, we will use the insulation workers data for estimating risks both in insulation work and in manufacturing. According to the Selikoff data, summarized in Table 7.9, the short-term, normally exposed worker under the 1982 Ontario control limit of 0.5 f/cc would be exposed to risks of 4.1 deaths per 1,000 workers, while the long-term, highly exposed worker would face risks of 16 deaths per 1,000 workers. The risks just mentioned are somewhat above the fatality risk in construction work, and above that in manufacturing.

We believe that these risks are unacceptably high. However, if the control limit were reduced to 0.1 f/cc, the estimated fatality risk would drop to 0.8 for the short-term, normal exposure, and 3.2 for the long-term, high exposure. We find that this health risk falls within the range of occupational health risks that have been found acceptable by society. No witness appearing before this Commission, however, has suggested that such low exposure levels could be achieved or measured reliably. We therefore recommend that:

7.14 ***The Government of Ontario should adopt a regulation which prohibits the use of amosite in Ontario manufacturing unless and until it is demonstrated to the satisfaction of the Ministry of Labour that there is a measurement technology that will satisfactorily detect low amosite concentrations so that a 0.1 fibre control limit can be enforced with a reasonable degree of confidence.***

As is the case with crocidolite, some workers will continue to be exposed to amosite where it has already been installed in buildings and must be disturbed. We believe that the respirator requirements for workers on construction projects should protect against breathing more than 0.1 f/cc of amosite. We therefore recommend that:

7.15 ***The Code for Respiratory Equipment for Asbestos on Construction Projects should provide for the use of respiratory protection that will ensure that the worker's exposure does not exceed 0.1 fibres per cubic centimetre where amosite asbestos is in the air.***

F.4 Insulation Work

Removing existing insulation, or performing other maintenance or renovation activities on existing buildings, may expose some workers to amosite fibres because of the presence of amosite in existing insulation. The risks these workers face can best be estimated using the Selikoff data in Table 7.9. These workers will be protected by the Regulation Respecting Asbestos on Construction Projects, which we discuss in Chapter 10. Recommendation 7.15 will specify the appropriate respiratory protection for these workers. In Chapter 10 we discuss the work practices we believe necessary to maintain the exposure of insulation workers and other construction workers at or below an acceptable level.

F.5 Cost of Recommendations per Life Saved

The risk assessment on which Table 7.9 is based can be combined with estimates of the cost of controlling workplace exposures to determine the incremental cost of life saving from adopting alternative control limits. It is interesting to compare these costs for three of our recommendations.

In the case of general chrysotile manufacturing, represented by friction products, the estimates of control costs by Dr. Gordon M. Bragg in his study for this Commission,[94] combined with the Table 7.9 risk assessments, suggest that moving from a 2 f/cc control limit to a 1 f/cc control limit involves a cost of about $68 million for every work-related death avoided.[95] This is a high cost when compared to the cost data discussed in Section E above, and even when compared with the occupational health and safety costs in that section.

In contrast, the health risks from textiles are so high that the marginal cost per life saved of reduced fibre levels is far lower than in friction products. Using the cost data from a study by the Research Triangle Institute (RTI) and the Table 7.9 health effects predicted from the Dement data, we calculate that reducing the control limit from 2 to 1 f/cc would cost about $0.66 million per life saved, and reducing it to 0.5 f/cc would cost $1.6 million per life saved, still far below the cost of life saving in brake manufacture.[96] To be consistent with the cost of life saving in brake manufacture would require far more strict controls in textile manufacturing, and perhaps a ban. We do not have costs for achieving an acceptable risk level in textile manufacturing, but it seems likely that prohibiting this manufacture as we have done would be no more costly per life saved than the 1 fibre control limit for brake manufacturing.

The risk assessment for asbestos-cement production with crocidolite is again very high, so that the cost per life saved of exposure reduction is low. Using the RTI cost data[97] and the Finkelstein-based risk assessment, the cost of reducing the control limit from 2 to 1 f/cc would be about $113,000 per life saved, while a reduction to a 0.5 f/cc control limit would cost about $60,000 per life saved. A control limit of 0.2 f/cc would cost about $1.1 million per life saved. Thus again, we appear justified by the cost figures in prohibiting the use of crocidolite in the manufacture of asbestos-cement products on the grounds that this is no more expensive as a way of saving workers' lives than the recommended controls on brake manufacture.

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[94] Gordon M. Bragg, *The Technical Feasibility and Cost of Controlling Workplace Exposure to Asbestos Fibres*, Royal Commission on Asbestos Study Series, no. 7 (Toronto: Royal Commission on Asbestos, 1982), Tables 4.2 and 4.5, reproduced as Tables 6.9 and 6.10 in Chapter 6 of this Report.

[95] Meeting a 1 f/cc control limit costs $1,350 per operator per fibre per year more than meeting a 2 f/cc control limit, or $13.5 million for 1,000 workers for 10 years. Table 7.9 shows that reducing the control limit by 1 f/cc saves 0.2 lives for short-term, normal exposure. The cost per life saved is thus $13.5 million divided by 0.2, or about $68 million.

[96] Research Triangle Institute, "Asbestos Dust Technological Feasibility Assessment and Economic Impact Analysis of the Proposed Federal Occupational Standard," draft report prepared for the U.S. Department of Labor, Occupational Safety and Health Administration, Research Triangle Park, North Carolina, September 1978. (Mimeographed.) The cost is $10.5 million for 1,000 employees, saving 16 lives, or about $0.66 million per life saved.

[97] Ibid. The cost is $8.7 million for 1,000 employees, saving 77 lives, or $113,000 per life, at a 1 f/cc control limit. Achieving 0.5 f/cc adds $2.4 million in cost, and 0.2 f/cc adds $28 million.

F.6 General

We have proposed regulations that distinguish among types of asbestos and that are particularly restrictive for specific manufacturing operations. We believe that the special hazards presented by textile operations result from the size distribution of fibres and perhaps their surface chemistry in the atmospheres of such plants. Even when the current membrane filter method of measuring airborne fibre levels reveals that a particular fibre level is achieved, the proportion of medically relevant long, thin fibres is greater in these plants than in other plants. If we knew for certain the toxicity of different fibre sizes and types, and the effects of surface chemistry, and if it were possible to measure these types and sizes and chemistry separately, then varied control limits could be specified that would provide equal protection in any plant using any type of asbestos. Our knowledge, however, is incomplete. The theories about the toxicity of fibre types, sizes, and surface chemistry are not precise. We have, therefore, relied on studies that have proved that certain situations have caused serious health problems in the past, and we have prohibited the recurrence of those situations directly. If at some time in the future the relationship between fibre type, size, and surface chemistry — and the resulting health effects — is sufficiently well developed that it is possible to achieve acceptable risk levels by specifying control limits alone, then the prohibitions on certain manufacturing operations should be replaced by the proper control limit.

Our recommendations above have been based on the hypothesis that asbestos fibres differ in their toxicity depending upon the size and perhaps the surface chemistry of the fibre. Because different types and manufacturing processes yield fibres that differ in their toxicity, then some of these types and processes should be subject to special restrictions. We recognize that these views are not held universally. However, the Dement and Finkelstein studies, which have emerged only in the last few years, compel the conclusion that health risks in different workplaces have differed greatly. The differences in estimated risk between the Dement and Finkelstein studies, on the one hand, and the J.C. McDonald and Berry studies, on the other, are so great that we feel confident in treating the risks of different fibre types and processes as truly different and regulating them accordingly. To apply a single control limit to all fibre types and processes would require that we ignore the Dement and Finkelstein studies, despite their being accepted in the scientific literature and to ignore almost all studies of the health effects of crocidolite. We find no reason to reject this evidence, and we are, therefore, logically led to recognize these observed differences in health risks in our recommendations for worker protection. With particular respect to differentiating among industrial processes for regulatory purposes, ours is, to the best of our knowledge, the first official study to frame formal recommendations to the effect that asbestos, in this instance chrysotile, requires different regulatory responses in different industrial processes.

How can employers be encouraged to reduce fibre levels below the numerical control limits that have been recommended? Under the *Occupational Health and Safety Act* there is a legal duty to reduce fibre exposure where practical, and this will presumably be encouraged by inspectors who can order a firm to reduce fibre levels even when they are in compliance with the 1 fibre control limit if in the Ministry's judgement this reduction is practical. In addition, the joint health and safety committee in any plant could work towards reducing fibre levels, even when the numerical control limit is met. Because we believe that compliance with this control limit will reduce asbestos risks to a level well below that of other risks faced by Ontario workers, we think it is appropriate that the joint committees share responsibility for deciding whether they wish to pursue health and safety by reducing fibre levels or by other means within a particular plant. The matter of enforcement is pursued in Chapter 8.

G. Other Fixed Workplace Exposure: Automotive Brake Mechanics

Approximately 40,000 automotive mechanics in the province of Ontario may be exposed in varying degrees to asbestos as a result of their work on brake maintenance and repair in 11,000 garages in the province. In Chapter 6 we note that this exposure may arise from two quite distinct sources: blowing dust from old drum brakes, and grinding or bevelling new brake shoes. The time-weighted average asbestos exposure caused by blowing dust from old drum brakes would, we conclude, generally be below 1 f/cc, in part because a considerable portion of this dust consists of material other than asbestos, and in part because most workers would spend only a few minutes per day actually blowing dust from brake drums. In contrast, grinding and bevelling of brake shoes can generate much higher dust levels, most of which consists of chrysotile asbestos. There are few shops in Ontario, however, where grinding and bevelling of new brake drums take place with any frequency.

In principle, all workers in automotive maintenance shops who may be exposed to asbestos dust as a result of brake repair are covered by the Regulation Respecting Asbestos since the shops would be places where ". . . asbestos is present . . . handled . . . and at which the worker is likely to inhale or ingest asbestos."[98] Thus, the 1 f/cc control limit for chrysotile contained in section 4(1) must be complied with. However, we consider it impractical to expect the 11,000 automotive shops across the province to conduct regular monitoring of the asbestos exposures of their mechanics. Nor would it be economical for the Ministry of Labour to perform such extensive monitoring, which would have to cover more than 15 times as many workers as are exposed to asbestos in manufacturing plants in the province. Fortunately, such monitoring is as unnecessary as it is impractical. Simple control procedures are identified in Chapter 6 that would bring the workers' asbestos exposure well below the 1 f/cc control limit. The situation of brake mechanics seems similar to that of construction workers, for whom regular air monitoring is not used, but instead protection is enforced by "regulation by procedure." In place of air monitoring, the Proposed Regulation Respecting Asbestos on Construction Projects (and our proposals for protecting workers from asbestos on construction projects in Chapter 10) specify procedures which must be followed to ensure worker safety without requiring the monitoring of air quality.[99] We believe that the same approach should be followed for brake mechanics.

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[98] Regulation Respecting Asbestos, s. 3(1).

While the exposure of brake mechanics to wear debris from blowing dust from old drum brakes will generally cause asbestos fibre levels below the 1 f/cc control limit, some control action may be desirable. Even if the wear debris is predominantly not asbestos, it still contains durable material of respirable size. Prudence would suggest taking some steps to control inhalation of this material. Furthermore, the Regulation Respecting Asbestos requires not only that the control limits be met, but that exposures be reduced to the lowest practical level. We conclude in Chapter 6 that the use of wet removal methods, in which the dust is gathered on a damp sponge, cloth, or brush, and essentially washed away, is both effective and economical. We believe that the use of wet removal methods is justified for old drum brakes. We therefore recommend that:

7.16 ***The Ministry of Labour, in applying the control limits of the Regulation Respecting Asbestos to automotive brake repair operations, should accept the use of wet methods of brake drum cleaning as prima facie evidence that the Regulation has been complied with.***

The high fibre concentrations observed during grinding and bevelling of brake shoes, in contrast to blowing wear debris from old brakes, and the low likelihood that this grinding has converted asbestos fibres into non-asbestos fibres, lead us to seek a more stringent protection regime for workers who perform these operations than is necessary for cleaning of brake drums. Workers engaged in grinding and bevelling of brake shoes may be protected by wearing a respirator or by attaching an exhaust ventilation system to the grinding machine. The appropriate procedure depends upon the situation in which the work is performed. In shops where grinding and bevelling are rarely necessary, it should be sufficient for the worker to use a respirator while grinding brake linings and to clean the grinding machine area regularly using wet methods. In shops where grinding and bevelling take place frequently, the extra cost of attaching a ventilation system to the grinding machine would be warranted by the considerable extra protection that this affords to the operator himself and to other near-by workers. We therefore recommend that:

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[99] Ontario, Ministry of Labour, Occupational Health and Safety Division, "Proposed Regulation Respecting Asbestos on Construction Projects and Related Codes."

7.17 ***The Ministry of Labour, in applying the control limits of the Regulation Respecting Asbestos to exposures of workers engaged in grinding and bevelling of brake drums in automotive brake repair operations, should accept as evidence that the Regulation is complied with:***

(i) that the operator is wearing a respirator while grinding and while cleaning the debris afterwards with wet methods, in shops where this operation occurs rarely; and
(ii) that the machines are equipped with local exhaust ventilation, in shops where grinding of brake shoes occurs frequently.

The above recommendations should solve the problem of the impracticability of regular air monitoring to determine compliance with the control limits specified in section 4 of the Regulation Respecting Asbestos. The remainder of the Regulation Respecting Asbestos, however, appears to apply to automotive repair shops and imposes a considerable burden on the large number of operations, each of which presents, as we have indicated above, modest worker exposures to asbestos. The Regulation can be interpreted to mean that every automotive repair shop must prepare an assessment of the likelihood of worker exposure to asbestos,[100] prepare an asbestos control programme, [101] and implement air monitoring and medical surveillance of workers.[102] We consider it eminently unrealistic to expect that this is being done, or will be done, in all 11,000 automotive shops across the province. Furthermore, we believe that it is not sensible to implement this elaborate machinery for what we have determined to be minor and isolated asbestos exposures.

We conclude in Chapter 6 that modest precautions will yield worker exposures well below the 1 f/cc limit, except that where grinding of brake shoes is performed frequently, exhaust ventilation is required. No purpose would be served by establishing the elaborate regulatory machinery in all these shops that is appropriate for manufacturing operations where substantial exposure may be experienced. All that is necessary is to encourage that minimal precautions be taken in these operations. We therefore recommend that:

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[100] Regulation Respecting Asbestos, ss. 6(1), 8, 9, 10.

[101] Ibid., s. 7(1).

[102] Ibid., ss. 7(2), 11, 12, 13, 14, 15, 16.

7.18 The Regulation Respecting Asbestos should be amended to specify that sections 6 through 16 do not apply to workplaces where the only exposure to asbestos arises out of the repair and maintenance of automotive brakes, unless grinding of brake shoes is performed frequently.

Research conducted by Commission staff in June 1981 revealed unfortunately that only a small fraction of the brake repair shops visited in Metropolitan Toronto treated brake dust generated from bevelling and grinding consistently with the procedures discussed above. In fact, in a number of locations we found that if any effort was expended in control, it was directed to controlling wear debris exposure via wet removal, while grinding and bevelling were undertaken with little or no protection. Training programmes for auto mechanics fail generally to convey the appropriate information about risks and worker protection. Considering the relative ease with which exposure to brake dust may be controlled, the problem of negligent removal seems to indicate not only a lack of available control equipment but an inadequate provision of information in the workplace as well. Since few of the workers involved in brake repair are organized, the task of informing workers of proper work practices falls onto both employers and the Minister of Labour.

The large number of brake repair shops, and the even larger number of mechanics who work on brakes, render difficult the problem of communicating the gist of the above recommendations to these workers. Fortunately, the risk faced by these workers is quite low, with the exception of those workers who regularly grind or bevel new asbestos-containing brake shoes. We therefore recommend that:

7.19 The Ministry of Labour should develop a modest information programme to communicate with the vast number of auto mechanics in Ontario the appropriate work practices as described in Recommendations 7.16, 7.17, and 7.18. A more intensive programme of communication should be designed to reach those mechanics working in brake specialty shops where, in particular, a considerable volume of grinding of new brake shoes is performed. The Ministry should also communicate appropriate practices to the secondary and post-secondary institutions in Ontario where auto mechanics training is currently provided.

Appendix to Chapter 7
Predicting Workplace Health Risks

Ronald J. Daniels*
and
Robin S. Roberts**

Section 1:
Dose-Response Models for Asbestos-Related Disease

A. Introduction

This Appendix outlines the rationale and mechanics of the model employed to predict the risks of asbestos-related disease presented in Chapter 7. Included in the Appendix is a complete set of results of the health effects simulations generated by making alternative assumptions concerning the characteristics of the workforce and the context in which their exposure is received. Specifically, we evaluate the impact of: (i) the duration and intensity of fibre exposure; (ii) population smoking habit; (iii) the age at which the population is first exposed to asbestos; and (iv) the frequency of population turnover, on the level of expected health risk.

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The authors would like to thank Mr. Julian Peto, now at the Institute of Cancer Research, Surrey, England, for extensive and helpful comments on an earlier draft of this Appendix. Authors of several studies discussed here, especially Dr. Murray M. Finkelstein, were helpful in responding to requests for clarification about their studies.

The Appendix is divided into three sections. Section 1 focuses on the development of dose-response functions specific to asbestos-related disease. These functions are then incorporated into a model which accounts not only for the gender, age, and smoking characteristics of the exposed population, but for their survival prospects as well. In Section 2 we review the various epidemiological studies conducted on populations occupationally exposed to asbestos and extract data from these studies that will enable us to extrapolate the health experience of historically exposed populations to future populations. Finally, in Section 3 we present the results of the simulations discussed above. Some of the health effects data that were used here are presented in the Appendix. Often, however, conclusions reached in Chapters 2 through 5 regarding the medical evidence will be referred to here, without full reference to the original sources. For a careful review of the medical evidence on health effects, see Chapters 2 through 5 of this Report.

B. Dose-Response Modelling and Asbestos-Related Disease

The first stage in calculating the future incidence of an occupational disease involves the construction of a dose-response function. The dose-response function relates the magnitude of a certain dose of a hazardous substance to the health effects that ensue. The measurement of dose is a complex matter and is discussed in Chapters 4 and 5 of this Report. Except when otherwise specified, we will represent the cumulative dose of asbestos as the product of intensity times duration. Since the behaviour of the dose-response function is based on data supplied by animal and epidemiological studies, deficiencies in these studies will also affect the quality of the resultant dose-response metric. For instance, epidemiological studies may be weakened by inaccurate measurements of dose and/or health response, while the results of animal studies may not extrapolate to man because of the physiological differences between humans and the species investigated.

Even if an epidemiological study is selected in which accurate measurements of dose and health response are found, the exposure of the study population may not span the full range of exposures that are now of interest. In fact, most worker populations that have been studied experienced past exposures to hazardous substances which were relatively large compared to current exposures. It is difficult to estimate a model from such data that is reliable for low doses.

To utilize the data furnished by a population previously exposed to a hazardous substance, a dose-response curve is fit to data which are characterized by many high-dose observations and few observations in a low-dose region. This step is accomplished tentatively at best, as there are a number of different dose-response models which could fit the mass of high-dose data with equal precision. Unfortunately, these competing dose-response functions yield very different results at low doses, which are of paramount importance for contemporary occupational health risk assessment.

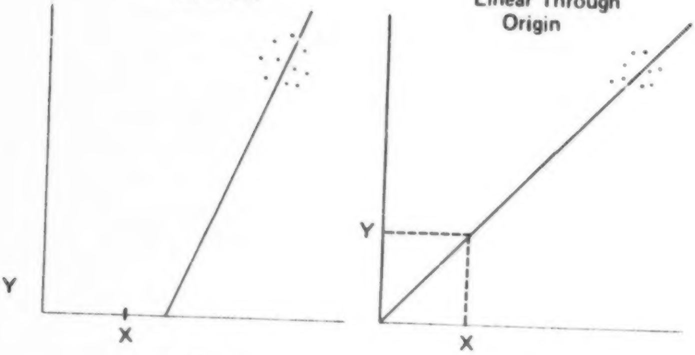
The dissimilarity in the behaviour of alternative dose-response functions at low doses means that the selection of one function over another will produce very different predictions of disease at these levels, as shown in Figure 1. Each of the dose-response functions depicted measures the cumulative dose of a hazard on the horizontal axis and response in terms of excess risk of mortality or incidence of disease on the vertical axis. Figure 1.A shows a threshold-response function. Below some limit value of dose there is no associated excess sickness or mortality. Figure 1.B illustrates a more frequently observed dose-response function that is linear through the origin. In this model a given increase in dose is matched by proportional increases in disease risk. Figures 1.C and 1.D depict dose-response models in which the response is a power function of the dose. In Figure 1.C (quadratic function) the response increases in proportion to the square of the dose, and in Figure 1.D (sublinear function) the response increases in proportion to a power of dose which is less than unity. Finally, Figure 1.E (cumulative normal) demonstrates the dose-response function in which response increases as if the distribution of susceptibility to a hazard was normally distributed. For a given low dose "X," notice that the magnitude of the predicted response "Y" varies significantly depending on which dose-response model is chosen: the risk predicted from dose "X" is greatest with the sublinear model, while the threshold model predicts that no health risk will result from this exposure.

What basis do we have for choosing the function best suited for modelling the future incidence of asbestos-related diseases? In the cases of lung cancer and mesothelioma, the available data are consistent with the assumption that excess mortality is proportional to the intensity of the exposure, although as Figure 1 shows, such consistency does not rule out other relationships. Most recent studies that have considered these data have chosen from the competing models a linear dose-response model as a reasonable compromise. The linear model is scientifically plausible, easy to work with, and conservative in that most other models would lead to lower, not higher, predictions of disease. We will use the linear model in the analysis that follows, for lung cancer and mesothelioma.

The decision not to enlarge our study to include predictions of other asbestos-related diseases was militated by a number of factors. Insofar as asbestosis is concerned, we have assumed that the amount of disease which would accrue from current exposures to asbestos would be negligible. This assumption is based upon the conclusion in Chapter 5 that clinical asbestosis is unlikely to occur at exposure levels that will prevail in the future. As well, we have not included predictions of other asbestos-related diseases

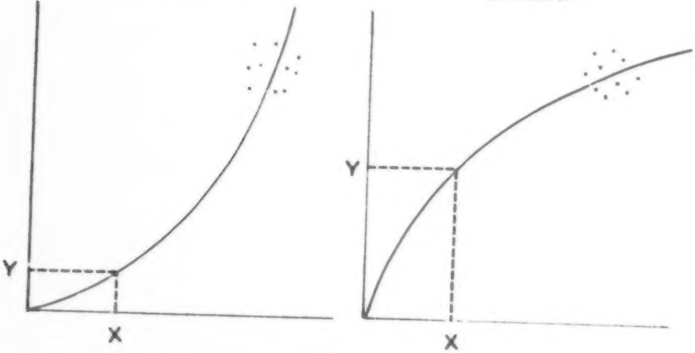
Figure 1

Alternative Dose-Response Curves Fitted to a Set of Observed Data Points



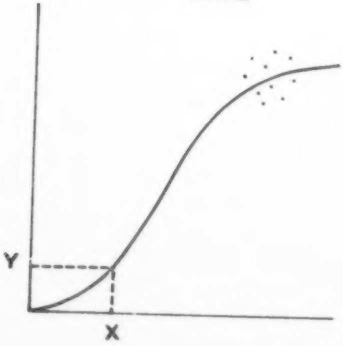
1.A Threshold

1.B Linear Through Origin



1.C Quadratic

1.D Sublinear



1.E Cumulative Normal

such as gastrointestinal and laryngeal cancer. Although a number of epidemiological studies have found elevated levels of mortality from these cancers among asbestos workers, the risk of experiencing these diseases has not been consistently demonstrated across all cohorts. Further, in the cohorts where excess rates of gastrointestinal and laryngeal cancer have been detected, the absolute amount of mortality from these diseases is much lower than the mortality from lung cancer, mesothelioma, and asbestosis. These factors, when combined with the uncertainty surrounding dose-response modelling for these diseases, have led us to exclude consideration of the effect of asbestos exposure on the levels of gastrointestinal and laryngeal cancer in our simulations.

C. Lung Cancer Dose-Response Function

C.1 The Measure of Lung Cancer Disease

Workers exposed to asbestos suffer an increased risk of lung cancer, but the disease also occurs in those not exposed to asbestos. To delineate between the amount of disease induced by exposure to asbestos and the amount of disease that would otherwise occur, the response metric of excess relative risk is employed.

The excess relative risk measure is obtained by comparing the health experience of a cohort exposed to a deleterious substance to the health experience of an unexposed cohort with similar population characteristics. The comparison yields a ratio of relative risk. By subtracting 1 from the relative risk value (to account for the background level of lung cancer), an excess relative risk measure is generated. The excess relative risk values derived from epidemiological studies will serve as the measure of the effect of asbestos on lung cancer incidence. The magnitude of this excess relative risk will vary from study to study because of: (i) different intensities and durations of exposure; (ii) varying fibre types and sizes; (iii) the personal characteristics of the population under study; and (iv) the differences in each study's design.

The health end point for which the excess relative risk measure will be derived is mortality from lung cancer. Since the time between diagnosis of lung cancer and death will rarely exceed 5 years, incidence of lung cancer and mortality from that disease may be treated as being virtually synonymous.

C.2 Selection of a Linear Dose-Response Function

The dose-response model we employ in calculating the future incidence of lung cancer is linear through the origin with respect to cumulative exposure to asbestos. Application of a linear function for estimating lung cancer risks from asbestos has been widely endorsed by members of the scientific community. For instance, Dr. William J. Nicholson, after discussing a variety of dose-response relationships and examining data gathered from epidemiological studies, concluded that the linear dose-response function is best suited for modelling asbestos-related lung cancer.[1] Dr. Nicholson found the alternative dose-response functions (log-probit, logit, multi-stage, one-hit, and threshold functions), developed for extrapolating results of animal data to human populations, deficient because the experimental data do not favour one model over another. Instead, Dr. Nicholson prefers the analysis developed by Dr. Kenny S. Crump et al. which finds that ". . . the added risk of cancer above 'background' from an external exposure is likely to be proportional to dose."[2] Further, Dr. Nicholson has stated that ". . . in the range of interest for occupational asbestos exposures, predictions from a linear extrapolation are indistinguishable from those of either the log-probit, logit, or any multi-stage model. . . ."[3]

Dr. Nicholson's conclusions, based on a survey of the theoretical literature, are buttressed by the findings reported in several epidemiological studies. These studies are discussed in depth in Chapter 5.

The adoption of a linear dose-response function with respect to cumulative exposure means that there is no greater weight attached to the effect of early as opposed to later exposure to asbestos. In this model, the health effects of a cumulative exposure of 30 fibres per cubic centimetre-years (f/cc-yrs) are the same regardless of whether the dose was received for 10 years at 3 fibres per cubic centimetre (f/cc) or 2 years at 15 f/cc. Also, since this model is linear through the origin, no exposure to asbestos can be considered "safe." Even relatively small exposures to asbestos will entail some additional risk of lung cancer.

C.3 Excess Relative Risk of Lung Cancer Independent of Age

The model used here assumes that the level of excess relative risk is solely a function of cumulative exposure and entirely independent of age at first exposure and pre-existing risk. This point is supported by the data presented in Figure 2 below. In this figure, the cohort studied by Selikoff,

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[1] William J. Nicholson, *Dose-Response Relationships for Asbestos and Inorganic Fibers* (New York: Environmental Sciences Laboratory, Mount Sinai School of Medicine of the City University of New York, 15 February 1981), pp. 9–13.

[2] Ibid., p. 11. See also, Kenny S. Crump et al., "Fundamental Carcinogenic Processes and Their Implications for Low Dose Risk Assessment," *Cancer Research* 36 (September 1976): 2973–2979.

[3] Nicholson, *Dose-Response Relationships for Asbestos and Inorganic Fibers*, p. 11.

Hammond, and Seidman [hereafter Selikoff] of insulation workers in the United States and Canada, first exposed to asbestos at ages 15 to 24, experienced a pattern of increasing relative risk that mirrors the pattern evident for workers drawn from the same cohort but first exposed to asbestos at ages 25 to 34.[4] While the slopes of the two functions are similar, the only difference between the two is their separation by a period of 10 years. This difference corresponds to the approximate difference between the mean age of first exposure of the two groups. While both curves in Figure 2 show a trail-off in disease incidence after 30 or so years, this feature is not incorporated in our model, for reasons discussed below.

C.4 Lag Between Onset of Exposure and Increase in Excess Relative Risk About 10 Years

Figure 2 also indicates that the time period between first exposure and manifestation of excess relative risk is fairly short. In the Selikoff cohort, a delay of about 10 years is experienced before an increase in relative risk is detected. A comparable graph from the study by Seidman, Selikoff, and Hammond, which also plots relative risk as a function of time since first exposure, reveals that the relative risk of developing lung cancer becomes elevated about 10 years after exposure commences.[5] See Figure 3. In order to capture this effect in our model, we will allow a delay of 10 years between the onset of exposure and the resulting increase in relative risk. Thus, the excess relative risk resulting from the first 10 years of exposure will be fully experienced at the end of the delay period. Thereafter, any increase in cumulative exposure will exert a direct impact on the response function.

C.5 Excess Relative Risk as a Function of Time

Another important feature of the model concerns the behaviour of the excess relative risk function with respect to time. The model assumes that after the 10-year period any increase in cumulative exposure will cause a similar increase in excess relative risk and will remain constant after total cumulative exposure is experienced. The reasonableness of presuming that excess relative risk of a given cumulative exposure to asbestos remains constant after cessation of exposure is somewhat problematic. Dr. Selikoff's epidemiological study of insulation workers found that the excess relative risk of lung cancer increased as cumulative exposure increased and then

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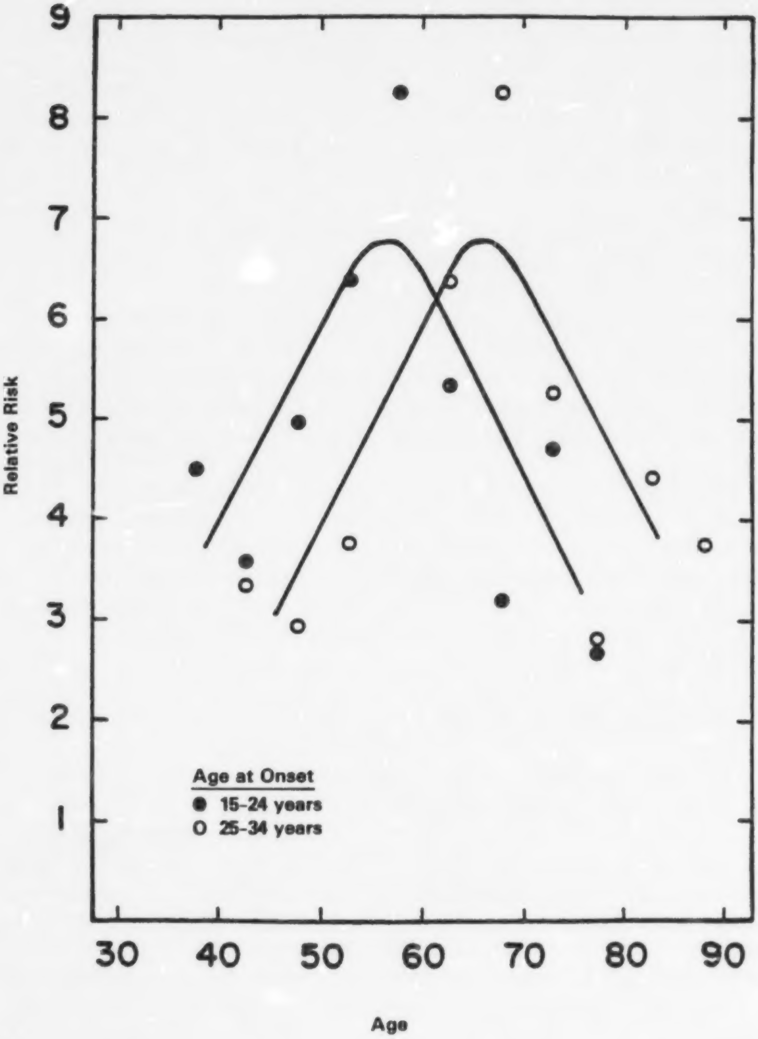
[4] See Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943–1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 91–116.

[5] See Herbert Seidman, Irving J. Selikoff, and E. Cuyler Hammond, "Short-Term Asbestos Work Exposure and Long-Term Observation," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 61–89.

Figure 2

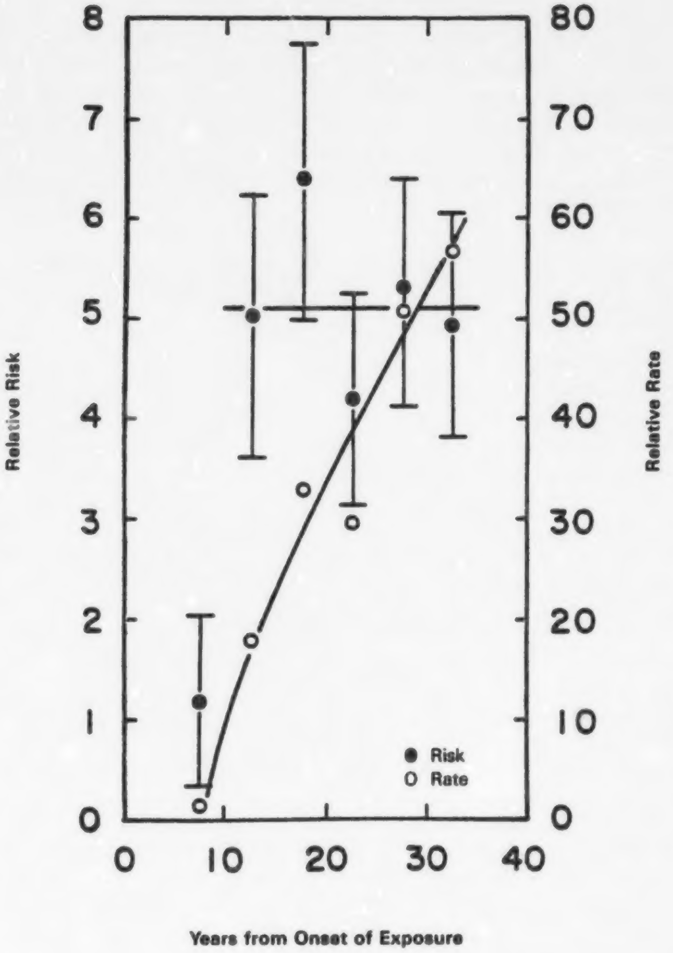
Lung Cancer Risk as a Function of Time Since First Exposure (Selikoff Study)



SOURCE: William J. Nicholson et al., "Cancer from Occupational Asbestos Exposure: Projections 1980-2000," in *Banbury Report 9: Quantification of Occupational Cancer,* eds. Richard Peto and Marvin Schneiderman ([Cold Spring Harbor, New York]: Cold Spring Harbor Laboratory, 1981), Figure 1, p. 89.

Figure 3

Lung Cancer Risk as a Function of Time (Seidman Study)



SOURCE: William J. Nicholson et al., "Cancer from Occupational Asbestos Exposure: Projections 1980-2000," in *Banbury Report 9: Quantification of Occupational Cancer*, eds. Richard Peto and Marvin Schneiderman ([Cold Spring Harbor, New York]: Cold Spring Harbor Laboratory, 1981), Figure 3, p. 92.

after remaining constant began to trail off after 30 years following onset of exposure. (See Figure 2.) It is likely that this trail-off in lung cancer relative risk may be related to the high past exposures experienced by the cohort; that is, workers who have had heavy asbestos exposure would suffer the risk of early removal from a competing asbestos-related disease such as asbestosis or mesothelioma. If some workers, whether for genetic or environmental reasons, are more susceptible to an asbestos-related disease than others it is conceivable that they would develop lung cancer early and be removed at that stage from the cohort. As a result, they would not survive to a point where the likelihood of developing an asbestos-related lung cancer was significant. Thus, removal via a competing cause of death would account for the trail-off in lung cancer relative risk as time since first exposure increases.

Although the trail-off effect was detected in the Selikoff cohort, most of the other epidemiological studies do not contain sufficient follow-up data either to reinforce or to refute the existence of this effect. In fact, in another epidemiological study with lengthy follow-up, Dr. Seidman and his colleagues were unable to find unequivocal evidence of a trail-off of excess relative risk with respect to time. Thus, motivated by the dearth of comprehensive epidemiological data which would allow us to define precisely the trail-off effect, we have refrained from building this effect into our model. Another reason to exclude a trail-off effect relates to our skepticism of the likelihood of substantial population removal by competing causes of death at low exposure levels. Recall that we have assumed that the level of asbestosis mortality (historically a major source of competition) accruing from current and future exposure levels will be negligible.

C.6 Lung Cancer Dose-Response Function Dependent on Background Lung Cancer Rate

To express response in terms of the excess lung cancer rate rather than excess relative risk, it is necessary to multiply the background lung cancer rate by the excess relative risk. This causes the response metric, previously a function solely of cumulative exposure, to be sensitive to the characteristics of the underlying population. That is because factors such as smoking, gender, and age which influence the level of lung cancer mortality in a population will exert a similar impact on the response metric when the product of excess relative risk and the underlying lung cancer rate is used.

However, reliance on a model in which excess relative risk is applied to the background lung cancer rate has important implications for the combined effect of smoking and asbestos exposure on lung cancer risk. Hammond, Selikoff, and Seidman found that smoking exerts a multiplicative effect on the lung cancer dose-response relationship.[6] This has been confirmed by a number of researchers and by this Commission, in Chapter 5. This means that if the excess relative risk caused by a given cumulative exposure is 5, the risk to asbestos workers who smoke becomes the background lung cancer rate for non-smokers multiplied by the relative risk of lung cancer entailed by smoking multiplied by 5. This point may be more easily understood by reference to Figure 4.

Our model predicts that in the absence of asbestos exposure and smoking, workers in the general population will experience a risk of lung cancer mortality that rises with age as curve A in Figure 4. For workers aged 20 years, the rate of lung cancer mortality is about 1,400 times lower than that experienced by workers aged 80 years. For unexposed workers who smoke, the lung cancer mortality curve shifts upwards by a constant factor. (See curve C.) Exposure to asbestos causes these curves to shift upwards by a factor determined by the product of cumulative exposure and the value of excess relative risk derived from the dose-response function. While this factor is the same for both smoking and non-smoking exposed workers, because the factor is applied to the smoking-specific background lung cancer rates, the magnitude of the final lung cancer rate for workers with equivalent cumulative exposures will differ in accordance with their smoking habit. (See curves B and D.)

The exposed population whose experience is simulated in this graph was first exposed to asbestos at age 20. Notice that even if we removed the delay constraint, the effect of exposure on lung cancer mortality at the earliest stages of their exposure is negligible. This is because in the years immediately following first exposure, the background level of lung cancer in the general population is quite low. Since the mortality rate is not significant at this stage, altering this rate multiplicatively by the appropriate excess relative risk value will not significantly affect lung cancer mortality.

When the actual mortality experience for a given cohort is plotted and stratified by age at first exposure, the same pattern that we have proposed for the model is evident. See Figure 5. Here, lung cancer mortality for smokers derived from Dr. Selikoff's insulation cohort is compared to an unexposed population. For workers first exposed at ages 15 to 24, the mortality curve is coincident with the mortality curve for the unexposed population until the late 30s. Thereafter, the mortality rates for exposed workers rise above the rate for unexposed workers by an amount dependent upon the intensity and duration of the exposure.

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[6] E. Cuyler Hammond, Irving J. Selikoff, and Herbert Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 473–490.

Figure 4

Predicted Lung Cancer Mortality as a Function of Age, Smoking, and Asbestos Exposure

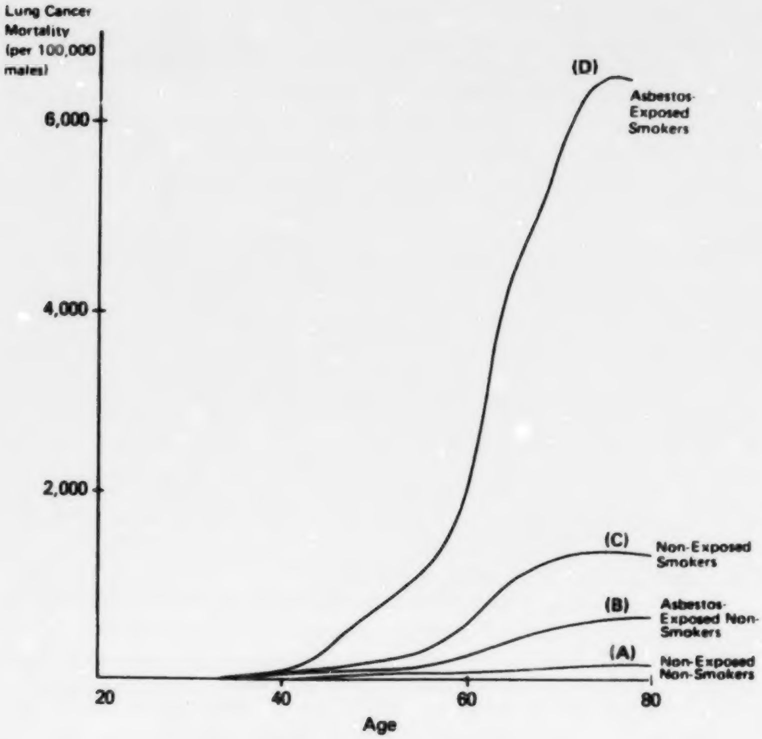
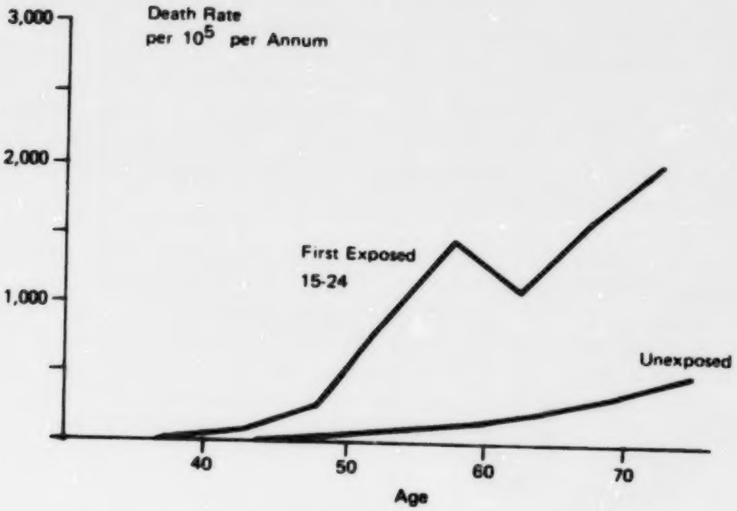


Figure 5

Lung Cancer Mortality Among North American Insulation Workers
(Cigarette Smokers Only)



SOURCE: Adapted from: Julian Peto, "Dose and Time Relationships for Lung Cancer and Mesothelioma in Relation to Smoking and Asbestos Exposure," in *Zur Beurteilung der Krebsgefahren durch Asbest* [Proceedings of the Bundesgesundheitsamt Asbestos Symposium], Berlin: February 1982, bga Schriften, MMV Medizin Verlag München, in press, 1983, Figure 6.

C.7 Combining Disease Rates with Survival Probabilities

The last theoretical issue to be resolved for our model concerns the age distribution of lung cancer mortality. Hitherto, we have dealt with lung cancer mortality in terms of disease rates. The mortality curves we have developed show that the rate of lung cancer mortality is highest when the population is oldest. However, these mortality rates must be viewed against the backdrop of the likelihood of an individual surviving to an age when these mortality rates are greatest. Since lung cancer is not the only source of death which an exposed population would experience, we must take into account the removal of members of the simulated population by death from other causes. So doing will cause the actual distribution of lung cancer deaths in an exposed cohort to fall below the levels we would expect without any natural removal.

Figure 6 clarifies this point, showing the yearly probability of death at different ages for a 20 year old male. If risk of lung cancer mortality is seen to increase as in Figure 6.A, this must be considered in the context of the survival curve drawn in Figure 6.B. A high risk of death from lung cancer at age 80 in Figure 6.A must be multiplied by the probability of surviving to age 80. Since it is likely from Figure 6.B that the worker will have died by some other cause before reaching that age, the actual probability of lung cancer mortality at age 80, which is shown in Figure 6.C, deviates from the level indicated in Figure 6.A.

However, as a result of asbestos exposure, the evaluation of the risk gradient at the higher end of the dose-response function will still be responsible for a great many lung cancer deaths in that range, and as a result the majority of lung cancer deaths related to asbestos will occur in older workers.

C.8 The Full Lung Cancer Model

From our preceding discussion, it is clear that any attempt to model a lung cancer dose-response function related to asbestos must be sensitive to the lung cancer mortality rate which would occur in the absence of exposure to asbestos. Ideally, such rates should be age-, gender-, and smoking-specific. In addition, it is important to account for worker survival patterns. The model we employ incorporates all of these variables. The number of excess deaths per year at age a in a cohort of size NOW first exposed to asbestos at age A is given by the following equation:

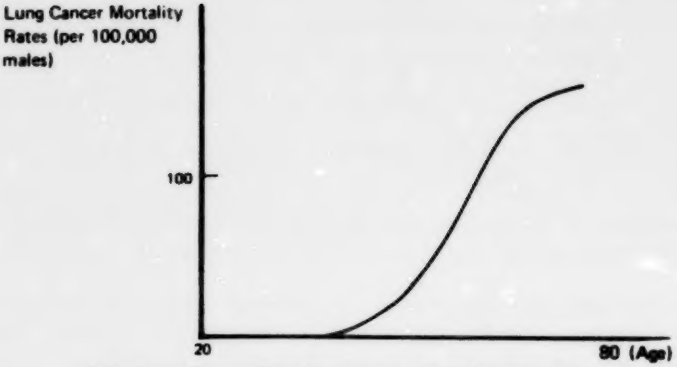
$$IE(a) = C*FCC*D*NOW*LU(a)*S(a)/S(A),$$

Where:

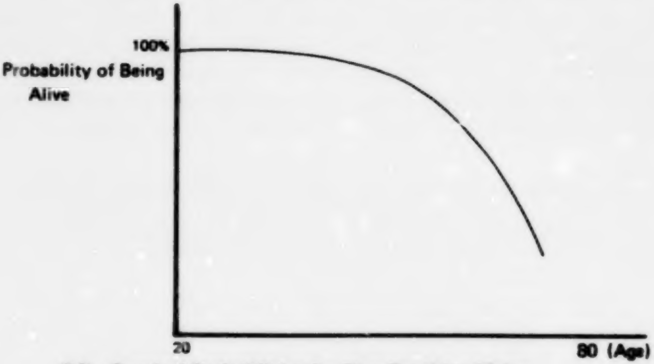
IE(a) = number of excess lung cancer deaths per year at age a resulting from asbestos exposure beginning at age A;

Figure 6

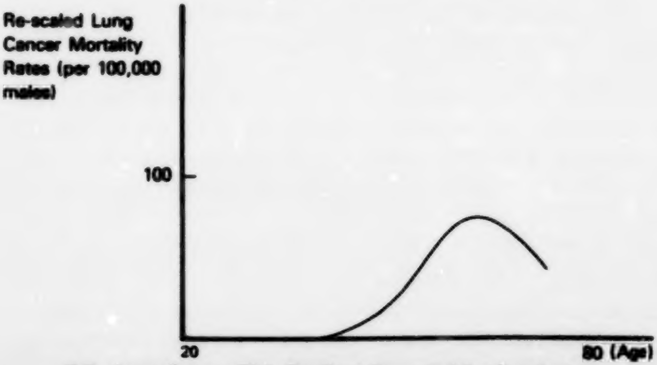
Yearly Probability of Lung Cancer Mortality



6.A Lung Cancer Risk Gradient for Non-Smoking Males



6.B Survival Probabilities for Non-Smoking Males



6.C Lung Cancer Risk Gradient Re-scaled for Survival

C = a proportionality constant derived from epidemiological studies which relates the cumulative dose of asbestos exposure to the excess relative risk of lung cancer;

FCC = average intensity of exposure to asbestos in f/cc;

D = duration of exposure to asbestos;

NOW = number of workers exposed;

LU(a) = the age-, gender-, and smoking-specific death rate for lung cancer;

S(a), S(A) = the cumulative probability of survival to ages (a) and (A) respectively.

The lifelong excess mortality of the cohort is the sum of IE(a) from a equals A to 80.

The input data are derived as follows: C values are derived from various epidemiological studies which investigated disease incidence among workers in various workplace settings. These studies are discussed in Section B below.

The age-, gender-, and smoking-specific lung cancer mortality data involve a rather complicated derivation. Since there is no central cancer registry in Ontario, we use Statistics Canada data on lung cancer mortality in Canada for 1978. These data are age- and gender-specific.

We further assume that the current mortality rates from lung cancer per 100,000 population will persist into the future. Whether or not the actual mortality rates in fact remain constant depends on several factors, including: the likelihood of improving the prognosis for lung cancer victims, the prevalence of environmental carcinogens, and smoking habits within the general population. Smoking habits are currently changing. For example, the percentage of females who smoke is increasing.

These lung cancer mortality data are modified so that the death rates would be smoking-specific (i.e., reflect the actual probabilities of survival experienced by smokers and non-smokers). This is accomplished by using the data furnished by the Health Promotion Directorate of Health and Welfare Canada on Canadian smoking patterns for the general population and weighting these proportions by a smoking lung cancer risk factor derived from the study by Richard Doll and Richard Peto of cancer rates among British doctors.[7] The information on Canadian smoking patterns is age- and gender-specific, while a risk constant derived from Doll and Peto of 10.4 is used for all ages and both sexes. This factor is the relative risk ratio that is obtained when lung cancer rates for smokers and non-smokers are compared.

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[7] Richard Doll and Richard Peto, "Mortality in Relation to Smoking: 20 Years' Observations on Male British Doctors," *British Medical Journal* 2:6051 (25 December 1976): 1525–1536.

Implicit in the calculation of our smoking-specific mortality rates is the assumption that all excess risks entailed by smoking at a certain age accrue only to those members who are still smoking at that age. This means that smokers who stop smoking, for example at age 50, will experience no increment in the lung cancer risk above that of non-smokers at later ages. To provide a more precise measure would require data on the percentages of smoking quitters in the general population, the age at which these occur, and the decrease in risk enjoyed by smoking quitters. Unfortunately, data of this specificity are not available. However, Doll and Peto have speculated that the decrease in risk from smoking quitters could be quite large. This hypothesis, if true, would enhance the validity of our model.

In order to account for the level of natural removal occurring during the life of an exposed population, survival rates were derived from life tables which were gender- and smoking-specific. The life tables were developed by using standard actuarial techniques and data on Canadian mortality rates for 1978. As for the lung cancer calculation, the general mortality rates were rendered smoking-specific by combining the percentages of smokers and non-smokers in the population with the relative risk of death for all causes entailed by smoking. These relative risk values are shown in Table 1.

Once modified, the mortality data are inserted into our model which calculates life expectancy tables. These tables determine, for a given population and mortality experience, the cumulative probability of surviving to a certain age and the amount of life-years lost for premature mortality. Because the data used to generate the tables are smoking-, gender-, and age-specific, so obviously are the survival rates. See Tables 2 and 3. These survival trends were assumed to remain constant for the lifespan of the exposed population.

Figure 7 summarizes the steps in calculating the risk of lung cancer mortality from asbestos exposure using the model just described.

D. Mesothelioma Dose-Response Function

Mesothelioma, in contrast to lung cancer, is a disease that is related almost exclusively to asbestos exposure. Mesothelioma is a cancer of the tissue covering the lungs and abdominal cavity (pleura and peritoneum). As with lung cancer, the prognosis for victims who develop mesothelioma is extremely bleak.

Table 1

Relative Risk of Mortality for Smokers

Age	Relative Risk of Death from All Causes
35-39	1.49
40-44	2.16
45-49	2.60
50-54	2.02
55-59	1.99
60-64	1.93
65-69	2.20
70-74	1.57
75-79	1.28

SOURCE: Adapted from: Richard Doll and Richard Peto, "Mortality in Relation to Smoking: 20 Years' Observations on Male British Doctors," *British Medical Journal* 2:6051 (25 December 1976): 1536 (Table XIII).

Table 2

Cumulative Survival Probabilities and Expected Life-Years Remaining for Smokers and Non-Smokers: Male

	Smokers		Non-Smokers	
Interval	Cumulative Survival Probability	Life-Years Remaining	Cumulative Survival Probability	Life-Years Remaining
20-24	1.000000	48.37	1.000000	55.01
25-29	0.990884	43.79	0.990884	50.50
30-34	0.983660	39.04	0.983680	45.85
35-39	0.9762299	34.47	0.9762299	41.18
40-44	0.9638647	29.78	0.9679144	36.50
45-49	0.9416987	25.42	0.9575451	31.87
50-54	0.904413	21.37	0.9427836	27.33
55-59	0.8486289	17.61	0.9135403	23.12
60-64	0.7686718	14.18	0.8692492	19.17
65-69	0.6607534	11.09	0.8038025	15.53
70-74	0.4697653	9.57	0.7088276	12.27
75-79	0.3339815	7.43	0.5711107	9.63
80-84	0.2129687	5.27	0.4027666	7.61

Table 3

Cumulative Survival Probabilities and Expected Life-Years Remaining for Smokers and Non-Smokers: Female

Interval	Smokers		Non-Smokers	
	Cumulative Survival Probability	Life-Years Remaining	Cumulative Survival Probability	Life-Years Remaining
20-24	1.00000	55.25	1.00000	61.12
25-29	0.99727	50.39	0.99727	56.28
30-34	0.99456	45.52	0.99456	51.93
35-39	0.99107	40.67	0.99107	46.60
40-44	0.98423	35.94	0.98648	41.81
45-49	0.97187	31.36	0.98072	37.04
50-54	0.94873	27.07	0.97167	32.36
55-59	0.91640	22.94	0.95514	27.98
60-64	0.87136	18.99	0.93127	23.53
65-69	0.80543	15.34	0.89407	19.40
70-74	0.68988	12.49	0.83339	13.63
75-79	0.57112	9.57	0.73907	12.31
80-84	0.44048	6.67	0.60360	9.51
85 +	0.25471	4.70	0.42801	7.39

Figure 7

Steps in Calculating Excess Lung Cancer Mortality from Asbestos Exposure

(i) Derive proportionality constant (C) which links excess relative risk of lung cancer with cumulative exposure.

(ii) Select a duration and fibre intensity for a hypothetical workforce.

(iii) Multiply duration by fibre intensity to obtain cumulative exposure.

(iv) Multiply cumulative exposure for workforce by proportionality constant (C) to determine the excess relative risk experienced in each quinquennium.

(v) Multiply the excess relative risk value by smoking-, age-, and gender-specific background lung cancer rates in each quinquennium to determine excess lung cancer rate.

(vi) Multiply the excess lung cancer rate in each quinquennium by the probability of being alive in that age interval.

(vii) For all age intervals, multiply the excess lung cancer rate (scaled to survival) by the number of workers exposed to asbestos. In determining the number of exposed workers alive in each quinquennium, take into account previous mortality from asbestos-related death.

(viii) Sum mortality over all periods.

D.1 Response Metric

The methodology employed for predicting the risk of mesothelioma mortality for an exposed population is different from that used to calculate lung cancer mortality. This difference arises from the absence of any relationship between the asbestos-related mesothelioma rate and the extremely low background rate of mesothelioma.[8] It is, therefore, not possible to calculate the mesothelioma rate as some multiple of the background rate. We will instead use mortality rates per year as the response metric for mesothelioma rather than relative or excess risk.

D.2 The Effect of Time Since First Exposure on Mortality

Another difference between lung cancer and mesothelioma dose-response functions concerns the pattern of risk after first exposure. Unlike the relative risk of lung cancer, which is solely a function of cumulative exposure, Mr. Julian Peto has found that the mesothelioma mortality rate is a function of not only the dose and duration of asbestos exposure, but also the time since first exposure.[9] As time passes after an initial exposure, the chance of death from mesothelioma occurring increases. Imputing consideration of time since first exposure into a risk equation causes early exposure to be weighted more heavily than subsequent exposure.

The relation between mesothelioma and time since first exposure has been found to be a power function; that is, if mesothelioma mortality for a population exposed to asbestos is plotted on a logarithmic scale against time since first exposure, a linear function is observed. Mr. Peto has found that the value of this exponent is between 3 and 4.[10] According to Mr. Peto, the added risk of mesothelioma mortality entailed by one day's exposure to asbestos at a given intensity is proportional to time since first exposure cubed. Integrating the risk curves over continuous exposure generates a risk curve depending on time since first exposure to the fourth. For intermediate exposure, that is, when time since first exposure is greater than duration, mortality is proportional to $T^4-(T-D)^4$: where T is equal to time since first exposure and D equals duration of exposure.[11]

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[8] Letter and attachments from Mr. Julian Peto, Imperial Cancer Research Fund, University of Oxford, Oxford, England to the Royal Commission on Asbestos, 21 September 1983.

[9] Julian Peto, "Dose And Time Relationships for Lung Cancer and Mesothelioma in Relation to Smoking and Asbestos Exposure," in *Zur Beurteilung der Krebsgefahren durch Asbest* [Proceedings of the Bundesgesundheitsamt Asbestos Symposium], Berlin: February 1982, bga Schriften, MMV Medizin Verlag München, in press, 1983, p. 2.

[10] Ibid., p. 3.

[11] Ibid., p. 4.

Figure 8 represents this relationship in diagramatic form. Here, each curve A through E shows the separate effects of successive occurrences of exposure to asbestos on the risk of mesothelioma mortality. Any point on an incidence curve represents the risk of mesothelioma at a specific time since onset of exposure for a given exposure interval. The risk of developing mesothelioma from the cumulative exposure is the sum of these curves. This total risk increases at a decreasing rate with further exposure, because the contribution to total mesothelioma incidence at time (T) of the most recent exposure curve is almost insignificant in comparison to the impact of the preceding curves. With risk growing at an increasing rate as a function of time since first exposure, in time the risk caused by early exposure is so high that the impact of subsequent exposure is dwarfed in comparison.

D.3 Pattern of Mesothelioma Dose-Response Function Independent of Age and Underlying Fibre Type

In our model, the pattern of mesothelioma risk is also independent of age at first exposure. Figure 9 shows the pattern of mesothelioma mortality of workers exposed at different ages plotted against time since first exposure. This exercise reveals parallel behaviour in the mortality pattern regardless of age at first exposure. For our purposes, it is important that a population exposed to asbestos in their mid-50s will not experience high rates of mesothelioma mortality. This is because mortality from mesothelioma will not become significantly elevated until at least 20 years after first exposure. In contrast, since the lung cancer risk function is dependent upon background lung cancer rates and these rates are highest for an elderly population, a population first exposed in their 50s will experience high rates of excess lung cancer once the delay period has been surpassed.

Mr. Julian Peto has also found that the pattern of mesothelioma risk predicted by the equation $T^4-(T-D)^4$ fits the pattern of mesothelioma mortality across a number of epidemiological studies *each with different underlying fibre types and fibre dimensions.*[12] Yet, while the pattern of mesothelioma risk is similar throughout a range of different studies, the absolute values of the pattern (i.e., the vertical intercept of the curve on logarithmic paper) may vary in accordance with fibre type and fibre process.

D.4 Mesothelioma Mortality as a Function of Time

Whether the mesothelioma risk continues to increase as a power function of time after 50 years of follow-up is not clear from existing studies.

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[12] Ibid., p. 3.

Figure 8

Risk of Mesothelioma Mortality Entailed by Asbestos Exposures as a Function of Time Since First Exposure

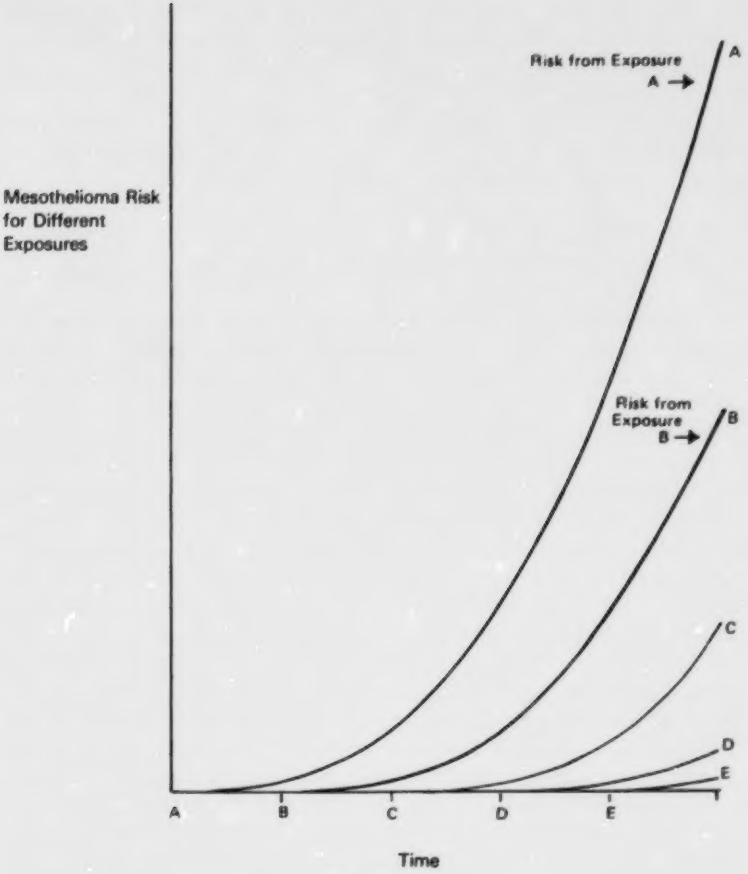
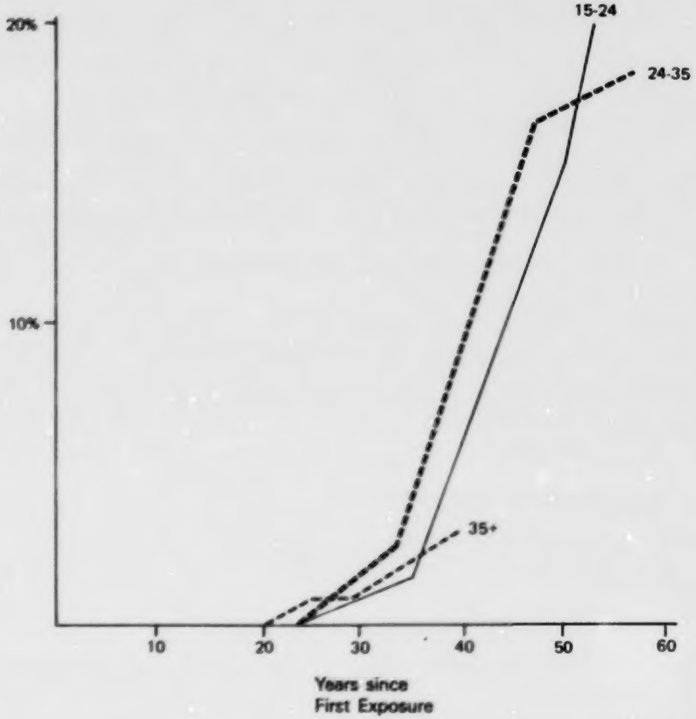


Figure 9

Cumulative Risk of Mesothelioma Mortality



SOURCE: Ontario, Royal Commission on Asbestos, Exhibit II-37, Tab 12, in RCA Transcript of Public Hearings, Evidence of Mr. Julian Peto, 29 July 1981, Volume no. 25(A), pp. 10ff: Mr. Julian Peto, Assorted Transparencies. (Mimeographed.) Based on data from Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 91-116.

Determining the behaviour of the mesothelioma risk function well after the onset of exposure is confounded by the same data limitations which hinder our understanding of the behaviour of the lung cancer function after lengthy follow-up. Again, we are reluctant to introduce in our model any trail-off for mesothelioma mortality after extensive follow-up because of the dearth of robust data which would enable us to model accurately this effect.

D.5 Presentation of Model

The relationships discussed above are integrated into Mr. Peto's mesothelioma model presented below:

$MI(a) = K*[T^4-(T-D)^4]*FCC*NOW*S(a)/S(A)$,

Where:

$MI(a)$ = number of mesothelioma deaths per year at age a in a cohort of size NOW resulting from asbestos exposure beginning at age A;

K = a proportionality constant derived from epidemiological studies which relates the risk of mesothelioma mortality to the intensity and duration of asbestos exposure;

T = time since first exposure = a-A;

D = duration of exposure to asbestos;

FCC = fibre intensity in f/cc;

NOW = number of workers exposed;

S(a), S(A) = cumulative probability of survival to ages (a) and (A) respectively.

The age-specific incidence of mesothelioma is more or less independent of smoking, but as non-smokers live longer than smokers it is still necessary to calculate separate life expectancy tables for smokers and non-smokers in the same way as for lung cancer. Non-smokers run a greater risk of developing mesothelioma than smokers, as a higher proportion survive to old age, when mesothelioma mortality is greatest. These life expectancy tables will be generated using the same data and techniques discussed with reference to lung cancer.

Figure 10 summarizes the steps in calculating the risk of mesothelioma mortality using the model just described.

E. Calculation of Mortality and Total Life-Years Lost

With the risk equations for lung cancer and mesothelioma that have

Figure 10

Steps in Calculating Mesothelioma Mortality from Asbestos Exposure

(i) Derive proportionality constant (K) which links rate of mesothelioma mortality with intensity and duration of exposure and time since first exposure.

(ii) Select a duration and fibre intensity for a hypothetical workforce.

(iii) For each quinquennium following exposure, multiply K by $[T^4-(T-D)^4]$ FCC to determine rate of mesothelioma mortality.

(iv) Multiply gender-, smoking-, and age-specific probability of being alive in each interval by number of exposed workers. Take into account removal of workers in previous interval by asbestos-related death.

(v) Multiply number of workers alive in each quinquennium by 5 years to determine total number of man-years in interval.

(vi) Multiply total number of man-years in interval by mesothelioma rate previously determined, to arrive at total mortality for interval.

(vii) Sum mortality over all periods.

been developed, we can now proceed to calculate both the total lifelong mortality risk and life-years lost from premature mortality for a group of asbestos workers. All that is required is that we establish a cohort of workers and specify their age, sex, and smoking characteristics along with the intensity and duration of asbestos exposure that they experience. From these data, the total number of deaths due to asbestos that will occur in the cohort is calculated by summing the numbers in each year [IE(a) and MI(a) in the equations in sections C.8 and D.5 of this Appendix] for each year of age from first exposure at age A to age 80. We selected 80 years for the outer boundary of the calculation because few epidemiological studies contained observations on the mortality experienced by workers surviving beyond this age.

This model includes the ability to determine the number of life-years lost from premature death. Once the total number of deaths due to asbestos at each age is known, this value is multiplied by the expected life-years remaining for this age. This index was developed from the survival probabilities outlined previously and displayed in Tables 2 and 3. Again, this calculation is executed for each year from first exposure at age A to age 80 and the results summed.

One last feature of our model is our removal of those population members who are expected to suffer an asbestos-related death in a certain quinquennium. This ensures that a worker who suffers early mortality will not be considered a part of the surviving cohort and thus will not be counted as dying twice from an asbestos-related disease.

Section 2:
Methodologic Review and Summary of Epidemiological Studies of Asbestos Exposure

Below we review the quality of seven epidemiological studies and then derive proportionality constants which will be inserted into our dose-response functions. Six of these studies are characterized by data on the conditions and intensity of exposure which were obtained for the workplace in which the cohort was exposed. The other study contains no exposure data, but can be supplemented by exposure estimates based on dust measurements in other places at other times. These data are used as surrogates for the experience of the cohort under study. We will first examine the six studies which contain environmental data from the workplace at which the cohort was exposed.

A. Review of Six Epidemiological Studies with Environmental Data

While there have been a multitude of mortality studies of asbestos workers, only six studies have been published which have associated environmental data of "reasonable" quality. None of these is entirely satisfactory in its own right and together they do not provide a precise estimate of the relationship between asbestos exposure and cancer risk. Thus, even though asbestos was "prescribed" in the United Kingdom in 1931 (admittedly because of asbestosis, not cancer), we must resign ourselves to the fact that the data relating dose to risk were still not definitive in 1983.

A.1 Review Technique

This section will briefly review the epidemiological quality of the six key studies and provide a summary of their methodology and results. The principal authors of the six studies are given below, along with brief identifying outlines of their work:

(i) J. Corbett McDonald et al. [hereafter J.C. McDonald] — study of miners and millers in Quebec;[13]

(ii) Vivian L. Henderson and Philip E. Enterline [hereafter Enterline] — study of retired asbestos products workers of the Johns-Manville Corporation;[14]

(iii) Julian Peto (et al.) [hereafter Peto] — study of asbestos textile factory workers at Rochdale, England;[15]

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[13] J. Corbett McDonald et al., "Mortality in the Chrysotile Asbestos Mines and Mills of Quebec," *Archives of Environmental Health* 22 (June 1971): 677–686. See also, J. Corbett McDonald et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910–75," *British Journal of Industrial Medicine* 37 (1980): 11–24.

[14] Philip E. Enterline, Pierre DeCoufle, and Vivian L. Henderson, "Respiratory Cancer in Relation to Occupational Exposures Among Retired Asbestos Workers," *British Journal of Industrial Medicine* 30:1 (January 1973): 162–166. See also, Vivian L. Henderson and Philip E. Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 117–126.

[15] Julian Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), pp. 829–836; and Julian Peto et al., "A Mortality Study Among Workers in an English Asbestos Factory," *British Journal of Industrial Medicine* 34 (1977): 169–173. See also, Geoffrey Berry et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory," *British Journal of Industrial Medicine* 36:2 (May 1979): 98–112; J.F. Knox et al., "Mortality from Lung Cancer and Other Causes Among Workers in an Asbestos Textile Factory," *British Journal of Industrial Medicine* 25 (1968): 293–303.

(iv) John M. Dement et al. [hereafter Dement] — study of asbestos textile factory workers at Charleston, North Carolina;[16]

(v) Geoffrey Berry and Muriel L. Newhouse [hereafter Berry] — study of the Ferodo friction materials factory workers at Derbyshire, England;[17]

(vi) Murray M. Finkelstein — study of asbestos-cement workers at Scarborough, Ontario.[18]

All six are conventional historical prospective studies. We have reviewed them methodologically in four areas: (i) study cohort; (ii) quantification of exposure; (iii) mortality ascertainment; and (iv) analysis. Each of these was subdivided into a number of features and a maximum numerical score assigned reflecting the perceived importance of that feature to overall methodologic quality. Finally, each study was assessed with respect to each feature and given a score (up to a maximum for that feature) to reflect how good the study was in this area. These scores are then summed up for the overall assessment of methodologic strength.

The results of this review are summarized in Table 4, and the methodologic details of each study are summarized in Table 5.

A.2 Methodologic Review

(a) Study Cohort

Selection Method (5 points) — We believe that the selection method least likely to result in bias is to select study subjects as they are first hired by the industry (an inception cohort), as opposed to cross-sectional censuses at one or more points in time. The Quebec study is clearly the best in this regard as the investigators identified everyone who had ever worked for one month or more and who was born between 1891 and 1920. The Rochdale and Charleston studies have adopted the more usual sampling procedure of sampling arriving employees but limited to those most recently hired when employee records were adequate. The study by Dr. Enterline of Manville employees selected study subjects at the point of retirement at age 65 and thus must be viewed much more skeptically because of intervening mor-

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[16] John M. Dement et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," *Annals of Occupational Hygiene* 26:1-4 (1982): 869-887.

[17] Geoffrey Berry and Muriel L. Newhouse, "Mortality of Workers Manufacturing Friction Materials Using Asbestos," *British Journal of Industrial Medicine* 40:1 (February 1983): 1-7.

[18] Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983. (Mimeographed.)

Table 4

Methodologic Assessment

Study Feature	Points	J.C. McDonald	Enterline	Peto	Dement	Berry	Finkelstein
A. Study Cohort							
Selection method	(5)	•••••	•	••••	••••	•••••	•••••
Inclusion/Exclusion	(2)	••	•	•	••	••	••
Sample/Complete	(1)	•	•	•	•	•	•
Size	(3)	•••	••	•	•	•••	•
Subtotal	(11)	11	5	7	8	11	9
B. Quantification of Exposure							
Work history detail	(3)	•••	••	•••	•••	•	••
Extent of industrial hygiene data	(5)	••	•	••••	•••	••	•
Quality of measures	(5)	•	•	••••	•••	••	•
Smoking data	(3)	•••	•		••		•
Subtotal	(16)	9	5	11	11	5	5
C. Mortality Ascertainment							
Quality of technique	(5)	•••••	••••	••••	•••	••••	••••
Completeness	(5)	••••	••••	•••	••••	•••••	••••
Availability of certificates	(3)	•••	••	•••	••	•••	•••
Length of follow-up	(2)	••	••	••	••	••	••
Subtotal	(15)	14	12	12	11	14	13
D. Analyses							
Suitability of external control	(3)	•••	••	••	•	••	•••
Internal comparison	(2)	••		••		••	••
Latency	(2)	••	••	••	••	•	••
Statistics	(2)	•	•	•	•	••	•
Exposure gradient	(2)	••	••	••	••	••	•
Bias discussion	(1)	•	•	•	•	•	•
Subtotal	(12)	11	8	10	7	10	10
TOTAL	(53)	45	30	40	37	40	37

Table 5

Methodologic Summary

Study Feature	J.C. McDonald	Enterline	Peto	Dement	Berry	Finkelstein
A. Study Cohort						
Selection method and criteria	All workers ≥ 1 month mines/mills Born 1891-1920	All Manville pensioners 1941-1967 (production workers only)	Hired 1933 or later ≥ 10 years service by 1972	White males ≥ 6 months service (≥ 1 month between 1/1/1940 and 31/12/1965	All employees hired between 1941 and 1960	All workers > 1 year of employment between 1948 and 1960
Size (dead)	11,379 (4,547)	1,075 (781)	679 (239)	768 (191)	13,460 (2,173)	535 (138)
Fibre type	Chrysotile	Chrysotile/ amphibole	Chrysotile/ some crocidolite	Chrysotile	Chrysotile/ some crocidolite	Chrysotile/ crocidolite
B. Quantification of Exposure						
Individual work histories	Yes	Yes	Yes	Yes	No	Yes
Industrial hygiene sampling method	Midget Impinger	Midget Impinger	Casella TP, high vol., membrane filter	Midget Impinger membrane filter	Membrane Filter simulations	Midget Impinger Membrane filter
Number of samples	4,152	Not reported	Routine since 1951	5,952	Routine since 1967	Routine since 1969
C. Ascertainment of Mortality						
Technique	Individual follow-up	SSA match and company records	NHS Central Registry	SSA and local agencies	NHS Central Registry	Personal follow-up and computer searches
Completeness	98% (post 1935)	Not reported	94%	97%	96%	94%
Available death certificates	97%	96%	Not reported	93%	100%	99.1%
D. Analyses						
Comparative population	Quebec	U.S.	U.K.	U.S.	England	Ontario
Latency assumed (since last exposure)	20 years	not used	20 years for dose-response	15 years	10 years	20 years
Exposure metric used for dose-response	mppcf	mppcf	f/cc-yrs	f/cc-yrs	f/cc-yrs	f/cc-yrs

tality. The resulting survival cohort is much more difficult to interpret. Mr. Berry studied all workers joining the company from 1940 onwards, when a new work history system was implemented. Recent hirings (after 1960) have not been included because of the relatively short time since first exposure. Dr. Finkelstein studied all employees since the plant's opening in 1948 who had accumulated one year or more of service and were employed initially prior to 1960, again for latency reasons.

Inclusion/Exclusion Criteria (2 points) — All studies, except the Berry study, applied a minimum duration of service criterion. They ranged from one month for the Quebec study to 10 years for Rochdale, with the Charleston and the Scarborough plants in between at 6 months and one year respectively. The Manville retirees study has an implied criterion since a man, to be included, must have been receiving a pension. From data in the paper the minimum duration of service would appear to be 3 years. Since there is particular interest in the dose-response function at low doses, a higher rating is assigned to studies that include exposures of short duration or low intensity such that the cumulative dose includes low as well as moderate or high levels.

Sample/Complete Enumeration (1 point) — All six studies included all eligible subjects as opposed to studying a sample and thus are equivalent in terms of sampling procedure.

Size (3 points) — The power of a mortality study to detect excess risk is directly related to the size of the study and in particular to the expected number of asbestos-related deaths. Mr. Berry studied 13,460 subjects, of whom 2,173 died during the follow-up period. This was the biggest mortality study of the six reviewed, although the Quebec study had more deaths with over 11,000 study subjects and 4,500 observed deaths. The Scarborough study (535 subjects), by contrast, was the smallest of the six. This is probably the single most important criticism, and yet the study does provide surprisingly extensive data on the risk of mesothelioma. While the other three studies are of the same order of magnitude in terms of size, the older cohort of Manville retirees had 4 times as many observed deaths as either Rochdale or Charleston, and is thus judged to be more powerful.

(b) Quantification of Exposure

Work History Detail (3 points) — The J.C. McDonald, Peto, Enterline, and Dement studies all had access to the individual work history of each study subject. While little information was provided as to the level of detail, all appeared comparable in their ability to describe where each subject worked. Even though individual job titles might be quite precise, each study has grouped them into dustiness "categories" for dose-response purposes. Dr. J.C. McDonald used thirteen categories in computing dose, while

Dr. Enterline used six. Dr. Dement and Mr. Peto both used sixteen categories.

Insofar as the Berry and Finkelstein studies are concerned, neither study has provided much background information on the degree of detail available in the work histories. There is a clear indication that this was a problem in the Berry study because the information was by cost centre rather than by job title. This necessitated a fair degree of judgement when interpreting a history. Thus, when Mr. Berry came to compute cumulative exposures he had to rely on company personnel to "guesstimate" where subjects had actually worked. Dr. Finkelstein provided little description other than saying that good quality individual histories were available. We are not told how many jobs had individual exposure estimates associated with them.

Extent of Industrial Hygiene Data (5 points) — Although quantity is no substitute for quality, the extent of industrial hygiene data in terms of the number of measurements taken and the years in which they were done is important.

While Dr. J.C. McDonald stated that over 4,000 midget impinger readings were available from annual surveys in the period 1949–1966, very few of these were in underground mines or open cast pits where the bulk of the cohort was exposed. The Dement study at Charleston had over 5,000 individual measurements in the plant during the period 1960–1975, but less than 200 measurements in each of the periods 1930–1945 and 1945–1960. However, this was judged to be a model plant by North American standards and, as such, probably has the best industrial hygiene data of any asbestos processor in the United States. The Rochdale study plant in the United Kingdom had routine monitoring from 1951 onwards and some *ad hoc* surveys before this date. While none of the publications indicated the exact number of measurements available, the plant had probably had the best U.K. data; hence its choice for the asbestosis study which was the basis for the 2 f/cc U.K. standard. Based on published descriptions, the Manville study would appear to have had much less exposure data than the Rochdale, Charleston, and Quebec studies.

The Berry study indicated that "routine" membrane filter measures were available since 1967 and personal sampling was introduced in 1968. Pre-1967 conditions were estimated by running the plant for a short period with equivalent pre-1967 ventilation levels and old production techniques. This is a valuable endeavour, but we are not told what data were collected and how long the simulation ran. Dr. Finkelstein listed dates of *ad hoc* government surveys and indicated that routine impinger data were available from 1960 onwards, and personal membrane filter estimates, from 1969. The extent of the data was not indicated. The calculations for dose-response purposes were based on 1969 and 1970 data, plus some crude assumptions about magnitudes of dustiness increases prior to 1969. No side-by-side impinger/membrane filter studies seemed to be available, so, unlike Dr. Dement's study, earlier impinger results have not been formally "converted" to equivalent fibre counts. Dr. Finkelstein made the point that the crude assumptions seemed to be in line with the trends in impinger counts, however.

Until concrete evidence shows the contrary, we conclude that the Finkelstein exposure data are about on a par with those of the Enterline study, and that the Berry study has slightly less extensive data than the Dement and Peto studies.

Quality of Industrial Hygiene Measures (5 points) — The quality of the industrial hygiene data is basically related to whether fibres have been measured and when the change from counting particles to fibres took place. The J.C. McDonald and Enterline studies are not strong in this regard since almost all of their industrial hygiene data were derived from the midget impinger as particle counts. Dr. J.C. McDonald did have some high volume data available for the mines in a survey done in 1968. Recent side-by-side surveys have been conducted in Quebec to try to develop a conversion factor for midget impinger to membrane filter fibre counts, but, because of the low efficiency with which the midget impinger collects fibres, these conversions have proved very unreliable. Both Dr. J.C. McDonald and Dr. Enterline thus continue to analyze dose-response relationships in terms of million particles per cubic foot (mppcf).

The switch to fibre counting, initially with long-running thermal precipitators and then membrane filters, commenced in 1961 at Rochdale, prior to which time Casella thermal precipitators were used to produce particle counts. At Charleston, midget impingers were used until 1971 with membrane filters introduced in 1965 and used solely from 1971 onwards. A total of 120 paired impinger/membrane filter samples were available, together with 986 measures of either impinger or membrane filter type taken during concurrent periods but not side by side.

The relationship between particle counts and fibre counts was studied carefully by Dr. Dement. The paired observations yielded a strongly significant correlation of 0.58 and indicated that 2.9 f/cc longer than 5 microns is equivalent to a particle count of 1 mppcf (the 95% confidence interval being 2.4 to 3.5). There was some evidence to suggest that the preparation area had a higher concentration of fibres. By comparison, the 986 concurrent samples yielded a conversion factor of 2.5 (the 95% confidence interval being 2.1 to 3.0) for most areas of the plant. The exception was, again, preparation which produced a conversion factor of 7.8 f/cc for each 1 mppcf. Subsequent calculations of dose-response relationships used conversions of 8 for preparation and 3 elsewhere in the plant.

We have dealt with this issue in some detail because it is one of the most contentious points raised in criticism of Dr. Dement's analysis. It is widely believed by experts that there should be a higher ratio of fibre to non-fibre particles in a textile mill than, say, in an asbestos mine or mill because processing is aimed at producing pure fibre. However, these conversion factors produced by Dr. Dement were almost equivalent to those arrived at by Dr. Graham W. Gibbs for the Quebec mines and a little lower than those for the Quebec mills. Thus, while somewhat lower than expected, Dr. Dement's values were derived from extensive data using valid statistical procedures and, in our view, must be taken at face value. Incidentally, early Casella thermal precipitator data at Rochdale were converted to fibre counts with a conversion factor which averaged at only 1 mppcf = 1.0 f/cc. The combination of earlier membrane filter data, plus measurements under simulated conditions, indicates that industrial hygiene data in the Berry study are of better quality than those in the Finkelstein study. Both would seem poorer in quality than the Dement data, which are in turn inferior to the Peto Rochdale data.

Smoking Data (3 points) — Clearly, with lung cancer as an outcome, smoking is an important confounding influence. Ideally, studies should have data on the smoking histories of each subject and an analysis of the effects of asbestos exposure conducted separately within smoking status groups (smoker, non-smoker, ex-smoker, etc.). An alternative, although a less satisfactory approach, is to demonstrate, usually on a sample basis, that the prevalence of smoking in study subjects is similar to the comparison population.

Of the six studies, the best smoking data by far were available in Dr. J.C. McDonald's Quebec study from a 1970 survey of live subjects and by interviews with relatives of men who had died prior to 1950. Dr. Dement's study at Charleston had smoking prevalence data available from a public health survey of the plant in 1964 and 1971. These rates were very close to the overall rates for U.S. white males in the same period. Dr. Enterline claimed to have smoking data from Manville's company records on about 25% of his cohort, of whom 77% were or had been smokers, again in line with national rates. Mr. Peto's study at Rochdale had very little to say about smoking except in relation to a small case-control type sub-analysis. The Berry study of the Ferodo plant at Derbyshire did not mention smoking data as being available. Dr. Finkelstein had smoking data on about 50% of his subjects at Scarborough.

(c) Mortality Ascertainment

Quality of Technique (5 points) — Dr. J.C. McDonald used the "best" technique of individually tracing each subject to locate the man or his relatives. Mr. Peto used the U.K. National Health Service (NHS) registry of a patient's current physician, which is highly reliable and constantly updated for mortality. Dr. Dement used primarily the files of the U.S. Social Security Agency (SSA) which records insurance payments to and from workers. Death information is thought to be incomplete in this file, but Dr. Dement complemented this search with local tax and postal information. Dr. Enterline used company records plus SSA files, which for a retired study group receiving a pension should be quite good. Mr. Berry determined the current status of his cohort by using the NHS physician register, which is of proven reliability. Dr. Finkelstein used a variety of techniques which seemed to involve personal follow-up but also included computer record linkage.

Completeness (5 points) — The five studies which provided figures on the success of follow-up all achieved well over 90%. The Rochdale and Scarborough studies were poorest with 94% each; and the Derbyshire (Ferodo), Charleston, and Quebec studies attained 96%, 97%, and 98% (after 1935) respectively. The study of Manville plants by Dr. Enterline did not provide a comparative figure but claimed the follow-up was perfect because the company would know if a pensioner had died. This seems a reasonable statement, but it is not clear if corroborative evidence was sought.

Availability of Death Certificates (3 points) — In the United Kingdom, death certificates are public documents stored centrally in Somerset House in London and are thus routinely available for study. In the U.S. and Canada, original certificates are stored in the administrative centre of the state or province of residence. This often makes it difficult to find certificates. While Dr. J.C. McDonald located 97% of death certificates, Dr. Enterline found 96%, and Dr. Dement, only 93%. The unknown causes of death cannot be included in cause-specific analyses and may bias the remaining causes of death if the likelihood of not finding a certificate varies with cause (perhaps lower for a compensable disease). Mr. Berry claimed that death certificates were available for all subjects found to have died, while Dr. Finkelstein located certificates for all but one deceased.

Length of Follow-up (2 points) — All six studies would appear to have had adequately long follow-up periods, usually from the 1930s to the mid-1970s. All had enough data to handle appropriately the issue of latency.

(d) Analysis

Suitability of the External Control (3 points) — The J.C. McDonald study used the Quebec mortality rates as the basis of its standard mortality ratio (SMR) calculations. Since two sites were involved and Quebec residents tend to stay in the province, this was a suitable choice. The Enterline study used all U.S. white males for comparison purposes. Again, since a number of plants in different locations were involved, this would seem reasonable compared to the alternative of a mix of states. The Peto study used the U.K. national rates in the Rochdale study and the Dement study used mortality rates from U.S. white males. Dr. Dement has come under some criticism for his choice of comparative population primarily because, in attempting an honest methodologic discussion of the main alternatives, he reported higher than average lung cancer mortality rates in the county around the asbestos plant. He attributed this to the presence of a large naval shipyard in the same county providing an alternative source of asbestos exposure especially during the war years when 29,000 men were employed.

Dr. Dement's decision not to use the local county rates is entirely appropriate; he might have used the state data instead, but since the lung cancer mortality rate in the state is virtually the same as the national rate, the latter is to be preferred. However, of more concern than the choice of external control is the possibility of contamination from the shipyard exposure prior to or after becoming a study subject by working at the asbestos textile factory.

Mr. Berry used mortality rates derived from the whole of England and Wales in calculating SMRs, whereas Dr. Finkelstein use the Ontario population. The latter approach would appear to offer a slightly better socio-economic matching.

Internal Comparison (2 points) — The Quebec, Rochdale, Derbyshire (Ferodo), and Scarborough studies all complemented the usual analysis of SMRs calculated with reference to an external control by an internal comparison. Dr. J.C. McDonald, Mr. Peto, and Mr. Berry did this by a case-control approach in which cases of lung cancer were matched with controls chosen from other study subjects alive when the case died. The comparison of exposure levels between cases and controls then forms the basis of the estimation of risk. Dr. Finkelstein used as controls a group of 87 men who worked in a rock wool process at the same plant.

Latency (2 points) — All authors were aware of the need to allow for an appropriately long latency period in calculating cancer risks from asbestos. Dr. J.C. McDonald and Dr. Finkelstein looked only at rates 20 years or more since first exposure; Dr. Dement chose 15 years; and Mr. Berry, 10. Mr. Peto had years since first exposure as a tabulated variable. Dr. Enterline did not specify a figure, but, presumably, since his study subjects had reached retirement, they had already got beyond the latency period. A specific verification of this would have been useful.

Statistics (2 points) — The Quebec, Rochdale, Manville, and Charleston studies did a comparable job at statistical analysis. None routinely reported exact values or, more importantly, provided confidence intervals on estimated risks. The J.C. McDonald study, unlike other studies, did not provide tests of significance on SMRs on philosophical grounds (presumably because the study group is viewed as a finite population in its own right and thus has no sampling variation). This study did, however, provide tests for dose-response relationships.

The statistical treatment by Mr. Berry was exemplary. Various dose metrics were discussed, including duration of exposure, cumulative dose, cumulative dose in the first 9 years, and a time-weighted metric. A confidencc interval was provided for the slope of the relative risk relationship with f/cc-yrs.

The Finkelstein study was adequate statistically, but the limited amount of data precluded subdivision of subjects beyond broad groupings. Even then, the numbers of deaths were very small and thus the associated risk estimates were imprecise. This imprecision would have been more obvious if the author had provided confidence intervals for the risk estimates.

Exposure Gradient (2 points) — Since these studies were selected from other cohort studies of asbestos workers because they provided good quality environmental data, it is not surprising that each had a detailed study of dose-response. The Quebec, Rochdale, Manville, and Charleston studies all showed strong gradients of risk with exposure both in terms of years of exposure and cumulative dust. Mr. Berry studied the nature of the relationship of lung cancer to cumulative exposure extensively. Because of relatively low average exposure in the cohort (only 5% of subjects had 100 f/cc-yrs or more), the overall excess mortality due to this cause with 100 f/cc-yrs of exposure did not reach conventional statistical significance.

Dr. Finkelstein's data suggest a linearly increasing mortality rate from mesothelioma with f/cc-yrs, but the relationship with lung cancer is much less clear. Both analyses were based upon small numbers so that the estimates of risk at defined exposure levels were less precise than those of the other two studies considered here.

Discussion of Bias (1 point) — All studies included discussion as to the possible biases inherent in their methodologic approach.

A.3 Summary of Methodologic Quality

From the bottom line in Table 4 it can be seen that all six studies score quite highly and would be judged by most scientists as "good" epidemiology, especially in the context of occupational health. Dr. Enterline's study would appear to be the weakest primarily because of a questionable sampling strategy and relatively poor exposure data. Dr. J.C. McDonald's study in Quebec is rated slightly above the remaining four, but all five are quite comparable in methodologic quality.

B. Comparison of Mortality Experience from the Six Studies with Environmental Data

The key results from the six studies are contained in Table 6. For all exposed subjects, the Berry study observed a relative risk for dust from lung cancer of 1.03 compared to 1.25 from the J.C. McDonald study, 2.7 from the Enterline study, 4.04 from the Dement study, and 5.12 from the Finkelstein study. The Peto study of the Rochdale experience has been puzzling because the relative risk for lung cancer in workers hired prior to 1951 was only 1.59 compared to 4.94 in workers hired after 1951. However, recent follow-up has shown a lower risk in the post-1951 cohort than was previously reported.[19]

Gastrointestinal cancer risks were mildly elevated in the studies by Drs. J.C. McDonald, Enterline, and Dement. The results from the Berry study indicated a negative relative risk for gastrointestinal cancer. However, in the Finkelstein study the relative risk from gastrointestinal cancer was significant, with a relative risk of 2.85.

The range of mesothelioma mortality levels was considerable. Dr. Enterline found only 5 cases of mesothelioma but suggested that many more cases occurred in the plant studied in men less than 65 and thus not eligible for the study. Dr. Dement included a systematic search for mesothelioma but found only one case. Ten deaths from mesothelioma were found in Quebec, 8 at Derbyshire (Ferodo), 8 at Scarborough (15 best evidence), and 8 at Rochdale.

Finally, one must be concerned about the competing risk of death from asbestosis or, more generally, non-specific fibrotic lung disease. Of the studies which reported these rates, all record substantial numbers of deaths from this cause which may have masked additional cancer cases. In each study, with the exception of Finkelstein, the number of deaths from pulmonary fibrosis was approximately equal to the number of excess deaths from lung cancer, so that, in the past at least, this has been an equally important end point. Mortality from lung cancer was almost twice the mortality from non-malignant respiratory disease in the Finkelstein cohort. From the Rochdale study, it would seem that the more recent risk of asbestosis is diminishing whereas lung cancer risk remains high. Mr. Peto warns this may be the effect of limited latency in a group hired since 1951. Furthermore, as noted above, more recent analysis has shown a lower lung cancer risk in the post-1951 follow-up.

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[19] E. Donald Acheson and Martin J. Gardner, *Asbestos: The Control Limit for Asbestos,* prepared for the U.K. Health and Safety Commission (London: Her Majesty's Stationery Office, 1983), paragraph 75, p. 12.

Table 6

Mortality Experience in Six Epidemiological Studies

Result	J.C. McDonald*			Enterline			Peto			Dement			Berry*, **			Finkelstein***		
a) Overall risks (≥ 20 years since first exposure)	O	E	RR§	O	E	RR	O	E	RR	O	E	RR	O	E	RR	O	E	RR
(i) Lung cancer	230	184.5	1.25	63	23.3	2.70	22 (Hired < 1950)	13.8	1.59	23	5.7	4.04	143	139.5	1.03†	21	4.1	5.12
							8 (Hired ≥ 1951)	1.62	4.94									
(ii) Gastrointestinal cancer	209	203	1.03	56	45.6	1.21	16	15.7	1.02	9	7.10	1.27††	103	107.2	0.96	8	2.8	2.83
(iii) Pleural mesothelioma deaths	10			5			7			0			8					
(iv) Peritoneal mesothelioma deaths	0			0			0			1			0			8 (15 best evidence)		
(v) Fibrotic lung disease deaths	42			31			8 (Hired ≤ 1950) 0 (Hired ≥ 1951)			151††			Not mentioned			9 (non-malignant respiratory disease)		
b) Lung cancer by dust/fibre level	mppcf-yrs	RR		mppcf-yrs	RR		Not reported			f/cc-yrs	RR	approx. mppcf-yrs	f/cc-yrs†	RR		f/cc-yrs	RR	
	< 10	1.0		< 125	1.98					< 27.4	2.23	< 9.1	50	1.03		< 30	2.31	
	10-99	1.2		125-249	1.80					27.5-109.5	3.57	9.1- 36.5	100	1.06		30-75	6.15	
	100-299	1.2		250-499	3.28					109.6-273	9.78	36.5- 91.3	150	1.09		75-105	12.08	
	300-599	1.8		500-749	4.50					≥ 274	15.53	91.3-182.6				105-150	9.0	
	≥ 600	2.9		≥ 750	7.78											≥ 150	2.89	

Notes: *These data are for the men only, not the entire cohort.
The J.C. McDonald cohort included 10,939 men (3,291 dead), and the Berry cohort included 9,113 men (1,787 dead).
**Ten years since first exposure.
***Production workers only.
§"O" means observed; "E" means expected; and "RR" means relative risk.
†Simulated exposure.
††Includes all deaths from first exposure.

C. Selikoff Study of Mortality Among Insulation Workers in the United States and Canada

As we mentioned at the outset of this chapter, Dr. Irving J. Selikoff's 1979 study of insulation workers in the United States and Canada did not contain any measurement of past exposure.[20] We feel that this deficiency is sufficiently grave to merit isolating the discussion of the Selikoff study from the six other studies which did include environmental data. The lack of accurate exposure data greatly detracts from the value of the study in providing proportionality constants for dose-response modelling. For this reason, we have refrained from undertaking a comprehensive evaluation of the study as we have done for the six other studies. Instead, we provide a brief review of the study.

The Selikoff insulation workers study documented the mortality experiences of 17,800 insulation workers who were enrolled in the International Association of Heat and Frost Insulators and Asbestos Workers in 1967. Dr. Selikoff and his colleagues observed 2,271 deaths between 1967 and 1976. The authors reported that prior to 1940, the subjects were exposed only to chrysotile asbestos and thereafter sustained exposure to both chrysotile and amosite asbestos during installation work.

The Selikoff study is characterized by comprehensive follow-up, robust data on death ascertainment, good information on smoking habits, and a particularly appropriate comparison population (white male age-specific U.S. death rates from the United States National Center for Health Statistics). Yet, these strengths are unable to compensate for the lack of environmental exposure data for the cohort.

Dr. Nicholson has attempted to provide exposure estimates which could be used as a proxy for the actual exposure experienced by the cohort.[21] The average estimate he arrived at for the cohort was based on recent measurements of asbestos exposure during insulation work. However, in order to account for both the reduced content of asbestos in the insulation products and the less frequent use of products containing asbestos during the period in which the measurement was taken, the average fibre intensity of 5 f/cc was multiplied by factors of 2 and 3 to generate estimates ranging from 10 to 15 f/cc. Only an average intensity can be used for the cohort, because there was not sufficient information available on the relative intensities of exposure each worker received. We assume an average exposure of 15 f/cc for the cohort.

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[20] Selikoff, Hammond, and Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943–1976."

[21] Nicholson, *Dose-Response Relationships for Asbestos and Inorganic Fibers*, p. 26.

The mortality experience of workers in the Selikoff study is presented in Table 7. The relative risk of lung cancer is 4.8 which is similar to the experience of the workers in the Finkelstein and Dement cohorts. Best evidence techniques reveal substantial mortality from mesothelioma: 170 deaths. In addition, the relative risk of other cancers is elevated to a factor of 1.5.

D. Derivation of Dose-Response Value from the Epidemiological Data

Having discussed and evaluated the methodological approach and results contained in seven epidemiological studies, we now proceed to derive dose-response values for lung cancer and mesothelioma from these studies.

D.1 Lung Cancer

We derive proportionality constants from each of the epidemiological studies discussed above by determining the ratio of the observed amount of lung cancer mortality to the cumulative exposure of the cohort. Ideally, the ratio or proportionality constant will be estimated by regressing the excess relative risk on cumulative dose, using a number of data points in a study. Each data point consists of the exposure and mortality rate for a subset of the cohort. However, where a satisfactory range of data points is not available, a single point representing the excess relative risk of mortality for the cohort and the corresponding average cumulative exposure will be used to produce the proportionality constant. This is considered a point estimate calculation. Since, by definition, the latter approach builds less information from the epidemiological study into the proportionality constant, the calculation is not as accurate as the former approach.

Once the proportionality constants have been derived, these values must be converted to a common dose metric to facilitate comparison. Since the generally accepted "active" component of the asbestos environment is a fibre (defined as having a length to width ratio of 3 to 1 or more, and a length of at least 5 microns) as opposed to non-fibrous dust, most standards are now specified in terms of fibres per cubic centimetre using optical microscopy and graticule counting techniques. (See Chapter 5.) The problem is thus to convert the particle counts from midget impingers used in earlier times to the equivalent fibre counts.

Unfortunately, all available side-by-side studies of the two methods suggest wide variability in conversion factors from plant to plant and job to job. In fact, it is not even clear that repeat observations over time in the same location provide a reliable conversion factor. The data available are

Table 7

Relative Risk of Excess Mortality from Asbestos-Related Disease in Insulation Workers Cohort

	Observed*		Expected	Relative Risk
	Death Certificate	Best Evidence		(Best Evidence)
Lung cancer	397	450	93.7	4.8
Gastrointestinal cancer	89	93	53.2	1.8
Pleural mesothelioma	—	61	—	—
Peritoneal mesothelioma	—	109	—	—
Other cancers	258	199	130.2	1.5
Fibrotic lung disease	177	204	53.8	3.8

Note: *Overall observed risk, 20 or more years after first exposure.

SOURCE: Adapted from: Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 108 (Table 16).

quite limited. As discussed earlier, Dr. Dement has incorporated a conversion factor of 3 f/cc equals 1 mppcf for most of his occupations based on U.S. public health survey data. These data were published separately by Ayer, Lynch, and Fanney in 1965 as part of a survey of four asbestos factories in the United States.[22] The overall mean conversion factor for the four plants was 9.5 f/cc per 1 mppcf, but it appears that the plant which Dr. Dement studied was lower at 5.2. This is quite close to Dr. Dement's conversion factor of 8 for fibre preparation and 3 for the remainder of the plant. Ayer, Lynch, and Fanney's average conversion is probably the best value we have available to convert the Enterline data into f/cc-yrs.

Dr. Graham W. Gibbs and Dr. Maurice Lachance presented, in their 1974 paper analyzing the Quebec mines and mills, side-by-side measurements of particles and fibres.[23] The data showed very little correlation and certainly wide variability in conversion ratios from location to location. In Quebec, a document prepared for the Comité d'étude sur la salubrité dans l'industrie de l'amiante, on reviewing all available data, recommended conversions of between 3 and 7 depending on location.[24] This range will be used to convert Quebec data.

The "raw" risk estimates in terms of exposure units of the original study reports are given in the bottom of Table 6. In four of the five studies which reported lung cancer mortality as a function of cumulative exposure, relative risk increases reasonably smoothly with exposure. However, for the Finkelstein study, the relationship is much less clear and the resulting linear model provides a poor fit to those data. This is probably due primarily to small numbers of subjects in each exposure group.

From these data, proportionality constants are calculated using regression techniques as discussed above. Of course, the uncertainty surrounding the proportionality constant derived from the Finkelstein data will be large considering the difficulty in fitting a straight line to the disparate response observations in that study.

For the Peto and Selikoff data, proportionality constants are calculated by using a point estimate rather than deriving the slope of the best fit line through a range of observations. For the Peto study, proportionality constants were calculated separately for the pre- and post-1951 cohorts and then compared. This exercise produced values of 0.002 and 0.013 for the pre- and post-1951 cohorts respectively. We have adopted a constant of 0.01 for the combined data, on the grounds that this value falls between the values for the two cohorts, assuming that both groups had roughly equivalent exposures of 300 f/cc-yrs. Recent follow-up shows a reduced risk in the post-1951 cohort, lowering the range within which the appropriate coefficient may lie. The appropriate constant may therefore be closer to 0.005 than to 0.01. On the other hand, it has also been suggested that the dust levels at Rochdale may have been overestimated, thus leading to underestimates of the dose-response relationship.[25] This would cancel out the overestimate of the post-1951 disease. We, therefore, use the coefficient of 0.01, recognizing that considerable uncertainty attaches to this value, as to many of the others derived here.

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[22] Howard E. Ayer, Jeremiah R. Lynch, and Julius H. Fanney, "A Comparison of Impinger and Membrane Filter Techniques for Evaluating Air Samples in Asbestos Plants," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 274–287.

[23] Graham W. Gibbs and Maurice Lachance, "Dust Exposure in the Chrysotile Mines and Mills of Quebec," *Archives of Environmental Health* 24 (March 1972): 189–197.

[24] See Michel Dagbert, *Études de corrélation de mesures d'empoussièrage dans l'industrie de l'amiante,* Document 5 for Québec, Comité d'étude sur la salubrité dans l'industrie de l'amiante (Beaudry Report), René Beaudry, J.C.P., Président (Montréal, Québec: l'éditeur officiel du Québec, October 1976). See also, J. Corbett McDonald and F. Douglas K. Liddell, "Mortality in Canadian Miners and Millers Exposed to Chrysotile," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 8.

For the Selikoff cohort, a single point estimate is used. The relative risk of lung cancer reported in Table 7 is 4.8 for workers with more than 20 years of follow-up. Converting to an excess risk, and dividing by 375 f/cc-yrs of exposure, yields a lung cancer coefficient of 0.0101. The exposure of 375 f/cc-yrs is based on an average exposure of 15 f/cc for 25 years.

The resulting lung cancer coefficients for all seven studies are presented in the top half of Table 8. The bottom half of Table 8 presents the estimated risk of lung cancer deaths at various levels of exposure based upon a linear model of excess risk. Note the range of values that is produced: the slopes derived from the studies by J.C. McDonald, Enterline, and Berry are roughly comparable at 0.000463 (at 3 f/cc conversion), 0.000693, and 0.00058 respectively. The Dement and Finkelstein slopes are highest at 0.0416 and 0.042 respectively. The almost identical values derived from point estimates in the Peto and Selikoff studies, 0.01, fall between the Dement and Finkelstein values, on the one hand, and the J.C. McDonald, Enterline, and Berry values, on the other.

Comparison of the risk of lung cancer at a cumulative exposure of 25 f/cc-yrs indicates that those studies which generated slopes with a low value not surprisingly predict only a slight increase in the relative risk of lung cancer, while the values derived from both the Dement and Finkelstein studies predict a doubling of the relative risk at 25 f/cc-yrs.

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[25] Acheson and Gardner, *Asbestos: The Control Limit for Asbestos*, paragraph 96, p. 15. See also, W.J. Smither and H.C. Lewinsohn, "Asbestosis in Textile Manufacturing," in *Biological Effects of Asbestos*, eds. P. Bogovski et al., IARC Scientific Publications, no. 8 (Lyon, France: International Agency for Research on Cancer, 1973), pp. 169–174.

Table 8

Estimated Relative Risk Coefficients for Lung Cancer

	J.C. McDonald		Enterline	Peto	Dement	Berry	Finkelstein	Selikoff
Coefficient Value C*	at 3 f/cc 0.000463 at 7 f/cc 0.000198		at 9.5 f/cc 0.000693	0.01	at 3 and 8 f/cc 0.0416	0.00058	0.042	0.0101
Estimated Risk	3 f/cc	7 f/cc						
25 f/cc-yrs	1.012	1.005	1.017	1.25	2.04	1.015	2.05	1.25
50 f/cc-yrs	1.023	1.010	1.035	1.5	3.08	1.029	3.1	1.50
75 f/cc-yrs	1.035	1.015	1.052	1.75	4.12	1.044	4.15	1.76
100 f/cc-yrs	1.046	1.020	1.069	2.0	5.16	1.058	5.2	2.01

Note: *The coefficient "C" may be inserted in the equation RR = 1 + C x FCC x D to estimate the relative risk of lung cancer from exposure to an asbestos concentration of FCC for a duration of D years.

D.2 Derivations for Mesothelioma Dose-Response Functions

Data on mesothelioma mortality from three of the studies discussed above are used to produce three estimated mesothelioma dose-response functions. Even though all seven epidemiological studies report some level of mesothelioma mortality, we are unable to calculate proportionality constants for all of them because not all studies present adequate mesothelioma mortality data. The three mesothelioma constants are all obtained from using regression analysis to fit the equation $MI(a) = K*FCC*D*[T^4-(T-D)^4]$ to the data shown in Table 9. We discuss below the derivation of the data used as inputs to the regression analysis for the Peto, Finkelstein, and Selikoff cohorts.

Peto's Rochdale Study — We combine the mesothelioma mortality of the pre- and post-1951 cohorts with the corresponding person-years of observation, in 5-year groups, to produce the data in Table 9. We assume that these workers experienced an average exposure of 30 f/cc for an average of 10 years.

Finkelstein's Scarborough Study — The mortality rates of each 5-year follow-up group are taken directly from the Finkelstein study. Data supplied to us by Dr. Finkelstein allow the determination that these workers were exposed, on average, to 5.25 f/cc for 10.5 years.

Selikoff's Insulators Study — The mortality rates of each 5-year follow-up group are taken directly from the Selikoff study. We have assumed, based on that study and other sources, that these workers were exposed to an average of 15 f/cc for 25 years.

Section 3:

Simulation of Predicted Disease

A. Introduction

With the values that have been derived for the lung cancer and mesothelioma dose-response functions, we can now simulate the effect of different exposure intensities on the level of mortality that will be experienced by a hypothetical workforce using the various constants from the seven studies analyzed above.

We assume that this workforce consists of 1,000 workers who are all exposed to the same level of asbestos. We also assume that 23% of the above workforce is female and 77% is male. In addition, we assume that

Table 9

Derivation of Mesothelioma Constants

	Peto* **(Rochdale)**	**Finkelstein**** **(Scarborough)**	**Selikoff***** **(Insulators)**
Assumed Exposure			
Intensity (f/cc)	30	5.25	15
Duration (yrs)	10	10.5	25
Time Since First Exposure	**Mesothelioma Mortality Rate (per thousand exposed workers)**		
10-14	0	0.4	0
15-19	0	0.4	0.15
20-24	0.43	2.7	0.29
25-29	1.26	6.3	1.55
30-34	2.39	9.6	2.76
35-39	3.94		6.29
40-44			6.33
45+			8.1
Derived Constant K	7.2×10^{-11}	213×10^{-11}	13.3×10^{-11}

SOURCES: * Julian Peto, "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," in *Biological Effects of Mineral Fibres*, vol. 2, ed. J.C. Wagner, IARC Scientific Publications, no. 30 (Lyon, France: International Agency for Research on Cancer, 1980), Table 1, p. 830.

** Murray M. Finkelstein, "Mortality Among Employees of an Ontario Asbestos-Cement Factory," Toronto, Ontario Ministry of Labour, February 1983, revised September 1983, Table 2. (Mimeographed.)

*** Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 109 (Table 17).

50% of the workers in the exposed population are smokers while the other 50% are not. Given that the average employment duration in three Ontario brake plants was found to vary between 3.8 years at one plant and 12.3 years at another, we have based most of our projections on an average employment duration of 10 years.[26] However, because exposure durations exceeding 25 years have been reported (e.g., at the Scarborough plant), we have used a duration of 25 years to represent long-term exposure. Lastly, the age at which work-related asbestos exposure began for the workforce is assumed to be 22. These assumptions concerning population characteristics will be maintained throughout the analysis unless otherwise indicated.

Our primary analysis will simulate the effects of reducing asbestos exposure levels. Several other analyses will also be performed. First, we will simulate the disease incidence in a workforce consisting only of non-smokers to contrast with our base workforce of 50% smokers. The interest in this analysis turns on the high risk of lung cancer sustained by asbestos workers who smoke and our model's ability to differentiate between the impact of asbestos on smokers and non-smokers. Second, we will examine the effects of varying the age of first exposure. This analysis is suggested by the impact of excess relative risk that rises slowly with age in both the lung cancer and mesothelioma dose-response function. Finally, we will simulate the effect of different rates of turnover in the workforce on the aggregate mortality of the workforce. Turnover will not affect lung cancer mortality but will affect mortality from mesothelioma, when the recruitment of new workers is among the young.

B. Changing Fibre Levels

In Table 10 we report the predicted asbestos-related mortality experienced by our hypothetical workforce at different fibre levels. The duration of exposure for this population is 10 years, and the age at first exposure is 22.

Noteworthy is the range of mortality evident at any given exposure level; for example, at 2 f/cc from 0.3 deaths and 3.9 life-years lost based on the J.C. McDonald study (at 7 f/cc conversion) to 295.4 deaths and 4,193.0 life-years lost from the values obtained from the Finkelstein study. These numbers differ by a factor of almost 1,000. Between these two extremes lies the mortality predicted by the Dement, Selikoff, and Peto studies with 62, 32.5, and 24.6 deaths predicted respectively at 2 f/cc. When the exposure is reduced by a factor of 10 to 0.2 f/cc, the mortality predicted by four of the eight studies is still in excess of 1 death per 1,000: Finkelstein (33.6); Dement (6.5); Selikoff (3.3); and Peto (2.5).

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[26] Sally Luce and Gene Swimmer, *Worker Attitudes About Health and Safety in Three Asbestos Brake Manufacturing Plants*, Royal Commission on Asbestos Study Series, no. 6 (Toronto: Royal Commission on Asbestos, 1982), p. 4.2. A recent study of three U.S. asbestos manufacturing plants reported that the average tenure of workers was 8.7 years, 7.6 years, and 9.2 years. See Alison D. McDonald et al., "Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant," *British Journal of Industrial Medicine*, in press, 1984, p. 6.

Table 11 presents estimates similar to those calculated for Table 1, but based on a duration of 25 years rather than 10 years. The relative levels of mortality risk are similar to those in Table 2. The predictions based on the Finkelstein study are highest, at a risk of 459.5 deaths for 2 f/cc, compared to the risk based on the J.C. McDonald study of 0.8 deaths.

By comparing the figures for a particular fibre level and a given study in Table 11 with the corresponding data in Table 10, the effect of increasing the duration of exposure can be observed. In the predictions in which only lung cancer mortality is calculated from the original study, the difference in mortality levels between the two tables reflects the linear impact of incremental duration on risk. For instance, at 1 f/cc the risk of mortality predicted by the Enterline study at 10 and 25 years is 0.54 and 1.35 respectively. This represents an increase in risk of death of 150% in response to an increase in duration of 150%. This is not merely coincidental; the 150% increase in duration causes a 150% increase in cumulative exposure, which causes a 150% increase in excess relative risk which acts on the background lung cancer rate. Thus, for these studies proportional increases in duration induce an exactly proportional increase in mortality. However, this relationship is somewhat less than proportional for the predictions based on the Finkelstein and Dement studies. This is because the lung cancer dose-response function is more powerful for these studies and causes a greater increase in the amount of asbestos-related mortality. As both the fibre level and the duration increase, the removal of workers suffering an asbestos-related death also increases, leaving fewer workers to face the mortality rate as the population ages. Therefore, when duration increases by 150% in the Dement study, mortality at 2 f/cc increases by only 132%.

In those predictions in which both lung cancer and mesothelioma mortality are calculated, an increase in duration is always matched by a less than proportional increase in mortality. This is attributable partly to the "removal effects" discussed above and partly to the behaviour of the mesothelioma dose-response function. We will defer a discussion of this latter point until the worker turnover analysis is presented.

C. The Effect of Exposing Non-Smokers Only

In Table 12 we present side-by-side calculations of the mortality that will occur in a workforce which is divided equally between smokers and non-smokers and a workforce containing non-smokers only. These calcula-

Table 10

Predicted Lifetime Mortality Risk for 1,000 Asbestos Workers Exposed to Different Fibre Levels for 10 Years*

Epidemiological Study	Fibre Level	Lung Cancer Mortality	Mesothelioma Mortality	Total Mortality	Total Life-Years Lost
Barry	2	0.9	–	0.9	11.3
[Derbyshire (Ferodo),	1	0.5	–	0.5	5.6
Friction Materials]	0.5	0.2	–	0.2	2.8
	0.2	0.1	–	0.1	1.1
Dement	2	62.0	–	62.0	782.1
(Charleston,	1	31.8	–	31.8	397.7
Textiles)	0.5	16.1	–	16.1	200.5
	0.2	6.5	–	6.5	80.6
Enterline	2	1.1	–	1.1	13.5
(Manville, Retirees)	1	0.54	–	0.54	6.7
	0.5	0.27	–	0.27	3.4
	0.2	0.11	–	0.11	1.4
Finkelstein	2	56.8	238.6	295.4	4,193.0
(Scarborough, Asbestos-	1	30.5	128.1	158.6	2,208.1
Cement Products)	0.5	15.8	66.4	82.2	1,133.4
	0.2	6.5	27.1	33.6	460.6
J.C. McDonald 1	2	0.7	–	0.7	9.0
(Quebec, Mining	1	0.4	–	0.4	4.5
@ 3 f/cc)	0.5	0.2	–	0.2	2.3
	0.2	0.1	–	0.1	0.9
J.C. McDonald 2	2	0.3	–	0.3	3.9
(Quebec, Mining	1	0.16	–	0.16	1.9
@ 7 f/cc)	0.5	0.1	–	0.1	1.0
	0.2	0.03	–	0.03	0.4

Table 10 (continued)

Predicted Lifetime Mortality Risk for 1,000 Asbestos Workers Exposed to Different Fibre Levels for 10 Years*

Epidemiological Study	Fibre Level	Lung Cancer Mortality	Mesothelioma Mortality	Total Mortality	Total Life-Years Lost
Peto	2	15.4	9.2	24.6	321.3
(Rochdale,	1	7.8	4.6	12.4	161.4
Textiles)	0.5	3.9	2.3	6.2	80.9
	0.2	1.6	0.9	2.5	32.4
Selikoff	2	15.5	17.0	32.5	431.3
(U.S./Canada,	1	7.8	8.5	16.4	216.8
Insulators)	0.5	3.9	4.3	8.2	108.7
	0.2	1.6	1.7	3.3	43.6

Notes: *Age at first exposure = 22; 385 male smokers; 385 male non-smokers; 115 female smokers; 115 female non-smokers.

Table 11

Predicted Lifetime Mortality Risk for 1,000 Asbestos Workers Exposed to Different Fibre Levels for 25 Years*

Epidemiological Study	Fibre Level	Lung Cancer Mortality	Mesothelioma Mortality	Total Mortality	Total Life-Years Lost
Berry	2	2.3	—	2.3	27.9
[Derbyshire (Ferodo),	1	1.1	—	1.1	14.0
Friction Materials]	0.5	0.6	—	0.6	7.0
	0.2	0.2	—	0.2	2.8
Dement	2	144.2	—	144.2	1,845.1
(Charleston,	1	76.4	—	76.4	961.2
Textiles)	0.5	39.3	—	39.3	490.7
	0.2	16.0	—	16.0	198.8
Enterline	2	2.7	—	2.7	33.4
(Manville, Retirees)	1	1.35	—	1.35	16.7
	0.5	0.7	—	0.7	8.4
	0.2	0.27	—	0.27	3.3
Finkelstein	2	127.1	332.3	459.5	6,410.7
(Scarborough, Asbestos-	1	72.0	187.5	259.5	3,500.0
Cement Products)	0.5	38.4	99.7	138.0	1,830.5
	0.2	15.9	41.4	57.3	752.5
J.C. McDonald 1	2	1.8	—	1.8	22.3
(Quebec, Mining	1	0.9	—	0.9	11.2
@ 3 f/cc)	0.5	0.5	—	0.5	5.6
	0.2	0.2	—	0.2	2.2
J.C. McDonald 2	2	0.8	—	0.8	9.5
(Quebec, Mining	1	0.4	—	0.4	4.8
@ 7 f/cc)	0.5	0.2	—	0.2	2.4
	0.2	0.1	—	0.1	1.0

Table 11 (continued)

Predicted Lifetime Mortality Risk for 1,000 Asbestos Workers Exposed to Different Fibre Levels for 25 Years*

Epidemiological Study	Fibre Level	Lung Cancer Mortality	Mesothelioma Mortality	Total Mortality	Total Life-Years Lost
Peto (Rochdale, Textiles)	2	37.7	14.0	51.7	658.4
	1	19.2	7.1	26.2	332.7
	0.5	9.7	3.6	13.2	167.2
	0.2	3.9	1.4	5.3	67.1
Selikoff (U.S./Canada, Insulators)	2	37.9	25.8	63.7	819.4
	1	19.3	13.1	32.4	414.5
	0.5	9.7	6.6	16.3	208.5
	0.2	3.9	2.6	6.6	83.7

Notes: *Age at first exposure = 22; 385 male smokers; 385 male non-smokers; 115 female smokers; 115 female non-smokers.

tions are based on an exposure intensity of 1 f/cc and a duration of 10 years. The level of lung cancer mortality is reduced by more than 75% in the non-smoking workforce based on all of the studies. Perversely, the level of mortality for mesothelioma actually increases in the non-smoking workforce.

This rather curious phenomenon occurs because eliminating smoking will not only lower the background levels of lung cancer, but will improve the likelihood of survival of the population. This is because non-smokers are generally healthier than smokers and live longer. Although the mesothelioma death rate is completely independent of smoking habits, the survival probabilities to which it is applied are not. Since increasing the proportion of non-smokers in the workforce will improve the survival prospects of the population, a higher proportion of the exposed population will live to an age when the risk of developing mesothelioma is greatest.

Noteworthy also in Table 12 is the relatively constant percentage change in the amount of mortality savings accruing from employing non-smokers only among those projections based on lung cancer mortality only. For the Berry, Dement, Enterline, and J.C. McDonald studies, mortality is reduced by 70 to 80% when mortality is calculated only for non-smokers. The mortality savings for the Peto and Selikoff projections are lower at 44% and 30% respectively because these projections include a substantial risk of mesothelioma. The mortality decrease for the projections based on the Finkelstein study is only 4% because of the very high mesothelioma risk in that study.

Finally, by examining the mortality projections for non-smokers based on those studies generating both lung cancer and mesothelioma dose-response functions, the importance of mesothelioma as the cause of death among non-smokers can be seen; death from mesothelioma is from 3 to 21 times more prevalent among non-smokers exposed to asbestos than is mortality from lung cancer in this cohort.

D. The Effect of Varying the Age of First Exposure

Table 13 demonstrates the reduction of mortality realized from the employment of older workers. For the projections based on the Finkelstein study, the decrease in asbestos-related mortality occasioned by increasing the age of the exposed population amounts to almost 78% of the mortality experienced by a population first exposed at age 22. However, the projections based on the Enterline study indicate only a 7.4% reduction in mortality for increasing the age of first exposure.

Table 12

The Predicted Effects of Smoking on Mortality Rates*

Epidemiological Study	50% Smokers and 50% Non-Smokers			100% Non-Smokers			Difference		
	Lung Cancer Mortality	Mesothelioma Mortality	Combined Mortality	Lung Cancer Mortality	Mesothelioma Mortality	Combined Mortality	Lung Cancer Mortality	Mesothelioma Mortality	Combined Mortality
Berry [Derbyshire (Ferodo), Friction Materials]	0.5	–	0.5	0.104	–	0.104	- 0.396	–	- 0.396
Dement (Charleston, Textiles)	31.8	–	31.8	7.3	–	7.3	-24.5	–	-24.5
Enterline (Manville, Retirees)	0.54	–	0.54	0.12	–	0.12	- 0.42	–	- 0.42
Finkelstein (Scarborough, Asbestos-Cement Products)	30.5	128.1	158.6	7.0	145.9	152.9	-23.6	+ 17.8	- 5.8
J.C. McDonald 1 (Quebec, Mining @ 3 f/cc)	0.4	–	0.4	0.08	–	0.08	- 0.32	–	- 0.32
J.C. McDonald 2 (Quebec, Mining @ 7 f/cc)	0.16	–	0.16	0.04	–	0.04	- 0.12	–	- 0.12
Peto (Rochdale, Textiles)	7.8	4.6	12.4	1.8	5.3	7.0	- 6.0	+ 0.7	- 5.4
Selikoff (U.S./Canada, Insulators)	7.8	8.5	16.4	1.8	9.7	11.5	- 6.0	+ 1.2	- 4.9

Notes: *Duration = 10; Fibre intensity = 1 f/cc; Age at first exposure = 22; Males = 770; Females = 230.

Table 13

The Predicted Effect of Increasing the Age at which the Workforce is First Exposed to Asbestos*

Epidemiological Study	Mortality Age = 22	Mortality Age = 52	Mortality Difference
Berry [Derbyshire (Ferodo), Friction Materials]	0.5	0.4	0.1
Dement (Charleston, Textiles)	31.8	29.0	2.8
Enterline (Manville, Retirees)	0.54	0.5	0.04
Finkelstein (Scarborough, Asbestos-Cement Products)	158.6	35.4	123.2
J.C. McDonald 1 (Quebec, Mining @ 3 f/cc)	0.4	0.33	0.07
J.C. McDonald 2 (Quebec, Mining @ 7 f/cc)	0.16	0.14	0.02
Peto (Rochdale, Textiles)	12.4	7.3	5.1
Selikoff (U.S./Canada, Insulators)	16.4	7.5	8.8

Notes: *Fibre intensity = 1 f/cc; Duration = 10 Years; 385 male non-smokers; 385 male smokers; 115 female non-smokers; 115 female smokers.

Again, the difference between the percentages is accounted for by the inclusion of mesothelioma mortality in the Finkelstein study. Since the amount of mesothelioma mortality is relatively low for the first two decades after exposure, increasing the age at first exposure of workers employed in the asbestos industry reduces the likelihood of surviving to an age where the risk of developing mesothelioma is greatest.

On the other hand, if the only mortality risk is from lung cancer, increasing the age of first exposure does not achieve any significant mortality reduction. To elaborate, increasing the age of the workforce reduces lung cancer mortality very little because lung cancer mortality is dependent upon the age-specific lung cancer rates for the general population. At age 62 (10 years after first exposure to account for the lag period), the rate of lung cancer mortality in the general population is substantially greater than the rate at age 32. Thus, increasing the age of the workforce increases the number of workers who are alive to suffer elevated levels of lung cancer risk.

With a different set of assumptions, the effect of this change could even become negative. We incorporate a 10-year lung cancer lag period in our calculations because we feel it is improbable that workers would experience an increase in the risk-gradient for asbestos-related lung cancer immediately upon exposure. However, if we allowed our workforce immediately to suffer increased levels of lung cancer upon exposure, the level of mortality from lung cancer could increase.

Finally, we examine the effect on mortality risks of reducing the age at first exposure in connection with exposure to very low levels of airborne asbestos fibre concentrations, to explore the risks to which building occupants might be exposed. Since building insulation is the substance to which much of the Selikoff cohort was exposed, we use the model derived from the Selikoff data as the basis for this simulation. An exposure of 0.001 f/cc is assumed, based on Chapter 9 of this Report. The lifetime risk of contracting a fatal disease from 10 years of exposure to 0.001 f/cc beginning at age 22 is 0.00002. The risk to a school child may be derived using the same model, but a younger age at first exposure. If the person is first exposed at age 7, and the exposure continues for 10 years, the lifetime risk of contracting a fatal asbestos-related disease is 0.000032, or almost double that of a 22 year old adult. Thus, according to these models, a child of 7 faces a risk of asbestos-related mortality from a given exposure in a building about twice that faced by an adult of age 22.

E. The Effect of Decreasing the Frequency of Worker Turnover

Consider the mortality that might result from exposing workers in 1,000 jobs for 20 years to 1 f/cc, under differing rates of labour turnover, with each worker experiencing his first exposure at age 22. If the same workers remain for only 4 years, there would be five cohorts with exposure durations of 4 years each. The predicted mortality for each cohort is shown in Column 1 of Table 14, and the total for the five cohorts is shown in Column 2. If workers remain for 10 years, there would be 2 cohorts with exposure durations of 10 years each. The mortality for each cohort is shown in Column 3 of Table 14, with the total for the two cohorts shown in Column 4. If the same workers remain for 20 years, there is one cohort with 20 years' exposure. The predicted mortality for this cohort is shown in Column 5 of Table 14.

As mentioned previously, if asbestos exposure caused only lung cancer, worker turnover would have little impact on the aggregate risk faced by the exposed population. However, when mortality from mesothelioma is calculated, differing lengths of duration will influence the levels of mortality that result. A comparison of Columns 2, 4, and 5 in Table 14 supports these propositions. The projections based on the Berry, Dement, Enterline, and J.C. McDonald studies, which include lung cancer only, show a negligible difference in mortality between five workforces exposed for 4 years and one workforce exposed for 20 years. Yet, the other projections, based on both mesothelioma and lung cancer mortality, show a significant difference between the amount of asbestos-related mortality predicted at different exposure durations. For example, mortality is reduced by 40% based on the Finkelstein study, 20% based on the Peto study, and 24% based on the Selikoff study as a result of reducing the number of workforces employed during 20 years from five to one.

Table 14

The Predicted Effect of Decreasing the Frequency of Worker Turnover

Epidemiological Study	1 Duration = 4	2 Mortality for 20 Years	3 Duration = 10	4 Mortality for 20 Years	5 Duration = 20
Berry [Derbyshire (Ferodo), Friction Materials]	0.18	0.9	0.5	0.9	0.91
Dement (Charleston, Textiles)	12.9	64.5	31.8	63.6	62.0
Enterline (Manville, Retirees)	0.22	1.1	0.54	1.1	1.1
Finkelstein (Scarborough, Asbestos-Cement Products)	78.1	390.3	158.6	317.3	235.9
J.C. McDonald 1 (Quebec, Mining @ 3 f/cc)	0.15	0.75	0.4	0.75	0.72
J.C. McDonald 2 (Quebec, Mining @ 7 f/cc)	0.07	0.35	0.16	0.32	0.3
Peto (Rochdale, Textiles)	5.4	27.0	12.4	24.8	22.0
Selikoff (U.S./Canada, Insulators)	7.4	36.8	16.4	32.7	27.8

Notes: *Fibre intensity = 1 f/cc; Age at first exposure = 22; 385 male non-smokers; 385 male smokers; 115 female non-smokers; 115 female smokers.

Chapter 8 Beyond Control Limits: Protecting Health in Fixed Place Industry

A. Introduction

Fundamental to our recommendation of control limits in Chapter 7 is the recognition that the Regulation Respecting Asbestos requires that "Every employer shall take all necessary measures and procedures by means of engineering controls, work practices and hygiene practices and facilities to ensure that the time-weighted average exposure of a worker to airborne asbestos is reduced to *the lowest practical level* . . . ."[1] (Emphasis added.) The Regulation goes on to specify a control level, but the "lowest practical level" is just as much a part of the Regulation as are the numerical control limits. While we have endorsed control limits for some exposure to asbestos, we recognize that compliance with these control limits does not eliminate all health risks to asbestos workers. We therefore attach major importance to reducing worker exposures to levels below the control limit, as required by the above-quoted language from section 4(1) of the Regulation Respecting Asbestos. This language enshrines in Ontario what we take to be a version of what is commonly referred to as the ALARA principle (As Low As Reasonably Achievable). A specific focus of this chapter will be on means of adhering to this principle. More generally, we are concerned with the enforcement and other aspects of the occupational health and safety system that have a bearing on the protection of worker health.

We perceive that problems of industrial health are particularly complex when compared to problems of industrial safety. In most cases, the dangers leading to industrial accidents are evident and the consequences immediate. Avoidance of the danger is frequently understandable and controllable at the shop floor level. Industrial disease, on the other hand, is primarily related to the environment in the workplace, which may be influenced by work practices but is largely beyond the control of the worker.[2] The worker may be unaware of, or at best only vaguely aware of, the existence of a hazard. The hazard may not be detectable by human senses; the consequences may not be evident for 10 or 20 years, or even until the next generation, and may strike randomly. For these reasons, occupational health is even more challenging than occupational safety in a setting where both place enormous requirements upon the many individuals and organizations involved and where complaints, particularly from labour unions and workers, have been numerous and pointed.

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[1] Regulation Respecting Asbestos, O. Reg. 570/82, s. 4(1), made under the *Occupational Health and Safety Act*, R.S.O. 1980, c. 321.

The *Occupational Health and Safety Act*[3] and its accompanying Regulation Respecting Asbestos establish and posit the existence of an Internal Responsibility System [hereafter IRS]. This system, through a variety of mechanisms, internalizes responsibility for workplace health to those most directly involved in the workplace: the employer, supervisor, and worker. While the IRS operates within the workplace, the Ministry of Labour provides both support for the IRS and its own enforcement functions under the Act through the activities of its Occupational Health and Safety Division.

In this chapter, we shall: (i) describe Ontario's occupational health and safety legislation with its focus on the IRS; (ii) sketch the role of the Ministry of Labour and its relation to the IRS; (iii) identify certain problems in the IRS and recommend some measures designed to deal with these problems; and (iv) assess the importance, in the particularly challenging realm of occupational health, of changes beyond the IRS that will help ensure the protection of workers.

B. The Ontario Setting: The Centrality of the Internal Responsibility System

On October 1, 1979, the *Occupational Health and Safety Act* was proclaimed in Ontario. This was a vital step in the evolution of workplace health and safety legislation, which began in the province in 1884 with *The Ontario Factories Act*.[4] At the time that the new legislation was passed, it replaced five acts operating in this field and administered by three government ministries. The *Occupational Health and Safety Act* consolidated all previous legislation.[5]

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[2] Ontario, *Report of the Royal Commission on the Health and Safety of Workers in Mines* (Ham Report), James M. Ham, Commissioner (Toronto: Ministry of the Attorney General, 1976), p. 16.

[3] *Occupational Health and Safety Act*, R.S.O. 1980, c. 321.

[4] *The Ontario Factories Act*, 1884, 47 Vict., c. 39.

An important catalyst in the creation of the new Act was the *Report of the Royal Commission on the Health and Safety of Workers in Mines* (the Ham Report).[6] A central theme of this document was that the system of internal responsibility should infuse the relationships among all participants in the workplace.[7] The main elements of the IRS would include an enhanced role for workers in dealing with workplace health and safety issues and a more continuous and accountable structure for problem-solving in this area. The major instrument for realizing these goals would be the joint health and safety committee. These principles became a cornerstone of the *Occupational Health and Safety Act* and the foundation for the regulations made thereunder. The Act created a new framework for organizing the various parties and responsibilities involved in protecting workers from both industrial accident and disease. Let us describe the elements of this Act and of the Regulation Respecting Asbestos which are central to this framework.

Section 41(1) of the Act gives the Cabinet authority to ". . . make such regulations as are advisable for the health or safety of persons in or about a work place." Section 41(2) of the Act goes on to enumerate possible regulatory areas, including the following:

> 11. respecting the reporting by physicians and others of workers affected by any biological, chemical or physical agents or combination thereof;
>
> 12. regulating or prohibiting atmospheric conditions to which any worker may be exposed in a work place;
>
> 13. prescribing methods, standards or procedures for determining the amount, concentration or level of any atmospheric condition or any biological, chemical or physical agent or combination thereof in a work place;
>
> 14. prescribing any biological, chemical or physical agent or combination thereof as a designated substance;
>
> 15. prohibiting, regulating, restricting, limiting or controlling the handling of, exposure to, or the use and disposal of any designated substance.

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[5] Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, pp. 10–25.

[6] Ontario, *Report of the Royal Commission on the Health and Safety of Workers in Mines*.

[7] Ibid., p. 6.

The Act defines a "designated substance" as ". . . a biological, chemical or physical agent or combination thereof prescribed as a designated substance to which the exposure of a worker is prohibited, regulated, restricted, limited or controlled."[8] To date, the Government of Ontario has designated six substances. It is in the process of designating six more and has given notice of four more to be designated.[9] Thus, the action of designating a substance triggers the regulation-making process,[10] with its attendant numerical control limits and codes for respiratory equipment, measurement, and medical surveillance.

The regulations reflect the Internal Responsibility System by delineating the powers and responsibilities of parties in the workplace to ensure the identification and control of designated substances. Furthermore, the Ministry of Labour has built its enforcement activities around the IRS. The routine, accident investigation, and compliance procedures of the Ministry aim to reinforce the effectiveness of the IRS in the workplace. More particularly, it is the Ministry's Occupational Health and Safety Division which has been given the role of propagating and supporting the operation of the IRS.

The essence of the IRS is that all those involved in the operation and conduct of a workplace should accept responsibility for the quality of its health and safety. In order for this system to be effective, the complete line of command, from the board of directors through the chief executive, senior officers and managers, supervisors and workers must accept the commitment to foster and improve health and safety in the workplace.

The IRS is embodied in the Act in a number of important ways, for example, in specifying the duties of employers, the responsibilities of workers for protecting themselves, and the joint duties of employers and workers in legally mandated health and safety committees.

B.1 Duties of Employers

Sections 14 and 15 of the Act set out a long list of duties for the employer. For example, section 14 states that the employer must ensure that all prescribed equipment, materials, and protective devices are maintained in good condition, and that they are used as prescribed. This section also requires the employer, *inter alia*, to provide information to the worker to protect health and safety; to afford assistance and co-operation to health and safety committees or representatives; to "take every precaution reasonable in the circumstances for the protection of a worker";[11] and to post ". . . a copy of this Act and any explanatory material prepared by the Ministry, both in English and the majority language of the work place, outlining the rights, responsibilities and duties of workers."[12] Section 15 gives additional duties to the employer, including the establishment of an occupational health service for workers; the keeping and maintenance of accurate records in respect of the use of biological, chemical, or physical agents; and the keeping, maintenance, and availability to the worker affected of his exposure records in respect of such agents. This section also imposes a duty on the employer to comply with any standard ". . . limiting the exposure of a worker to biological, chemical or physical agents as prescribed."[13]

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[8] *Occupational Health and Safety Act*, s. 1, par. 6.

[9] The six designated substances are asbestos, coke oven emissions, lead, mercury, isocyanates, and vinyl chloride. The six for which there are proposed regulations are acrylonitrile, arsenic, benzene, formaldehyde, noise, and silica. The Government of Ontario has given notice under s. 41(2), par. 14 of the Act that it intends also to regulate four other substances, namely, cadmium, chromium, ethylene oxide, and styrene.

[10] *Occupational Health and Safety Act*, s. 41.

B.2 Duties of Workers

The Act requires the worker to take responsibility for his own health and safety in the workplace. A worker's duties, as set out in section 17 of the Act, include conducting his work in compliance with the provisions of the Act and using the equipment, protective devices, or clothing that the employer requires. Moreover, the worker is required to report to his employer or supervisor the existence of any hazard; the absence of, or defect in, equipment or protective devices; and any contravention of the Act or regulations made thereunder. Thus, every worker in effect becomes a front-line health and safety inspector.

Section 23 carries the worker's front-line inspector role beyond that of merely reporting hazards and contraventions to the employer or supervisor. Section 23(3) gives the worker the right to refuse to do work which he has reason to believe is likely to endanger himself or another worker. This provision may be used not only in respect of any specific equipment, machine, or device, but also more broadly in respect of the physical condition of the workplace. A worker who chooses to exercise this right of refusal has a duty to report the circumstances of his action promptly to his employer or supervisor and, if possible, to a worker representative of the health and safety committee, who thereupon must investigate the situation. If, after investigation and any necessary action, the worker still considers the situation to be dangerous, he may continue his refusal, whereupon a Ministry of Labour inspector must be called in. During both the initial in-house investigation and any subsequent inquiry and decision by the Ministry inspector, section 23(11) stipulates that "... no worker shall be assigned to use or operate the equipment, machine, device or thing or to work in the work place or the part thereof which is being investigated unless the worker to be so assigned has been advised of the refusal by another worker and the reason therefor."[14]

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[11] Ibid., s. 14(2)(g).

[12] Ibid., s. 14(2)(h).

[13] Ibid., s. 15(1)(g).

B.3 Joint Responsibility

The investigation of unhealthy or unsafe work conditions is carried out, in the first instance, by representatives of labour and management together. Such investigation is one of several joint activities that are central to the principle of the IRS. Section 8(2) of the Act mandates the establishment, in workplaces of twenty or more workers, or in any workplaces where a designated substance is present, of a joint health and safety committee. That the committee structure is critically important to the IRS and to Ontario's health and safety system is signalled several times in the Act. First, the Act repeatedly stipulates that in all significant workplaces there must be an institutionalized concern for identifying situations that may be a source of danger or hazard to workers, recommending investigation or specific courses of action, and following-up to ensure that appropriate attention is given to any situation. Second, section 8(5) of the Act states that the joint committee must consist of at least two persons, of whom at least half must be workers; such worker representatives should be selected by the workers or, where applicable, by the relevant union(s). Here the evident intention of the Act is to institutionalize the concept that workers are equally responsible for health and safety along with management. Third, section 8(6) gives the committee "... power to ... obtain information from" employers concerning the identification of potential or existing hazards, and health and safety experiences and practices in similar or other industries. Fourth, section 8(8) gives the worker members of the health and safety committee the power and duty to designate one worker representative to inspect the physical condition of the workplace once a month.[15] Fifth, the joint health and safety committee provides a vehicle whereby Ministry of Labour inspectors get a view of the issues of concern to workers because it is envisaged that a worker representative will accompany an inspector on his plant tour or, absent such a representative, that the inspector shall consult workers during his tour.[16] Sixth, a copy of any orders or reports issued by an inspector must be lodged with the health and safety committee and must furthermore be posted conspicuously in the workplace.[17] The committee is thus a vehicle for ensuring follow-up to orders issued during an inspection, and for informing workers at-large about the conditions of their work environment. Finally, the Act states that members of health and safety committees are entitled to take the necessary time from work in order to attend committee meetings and perform prescribed inspection duties. The time so spent is to be considered paid work time, thereby once again indicating that workers' concerns for health and safety are an integral part of daily work activity.[18]

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[14] Ibid., s. 23(11).

[15] These inspections can be carried out more frequently in relation to a critical injury or where a director of the Ministry of Labour so directs. See *Occupational Health and Safety Act*, ss. 8(9) and 23(4).

[16] Ibid., ss. 28(3) and 28(4).

[17] Ibid., s. 29(6).

We note that the recommendations of joint health and safety committees are advisory rather than binding; the long-run effectiveness of the IRS therefore hinges upon the quality of the response to this advice.

B.4 The Internal Responsibility System and the Designated Substances Regulations

Having incorporated the concept of internal responsibility in the Act through the establishment of such institutional arrangements as the joint health and safety committee, the government uses the IRS foundation to ensure compliance with the regulations promulgated for the control of designated substances. These regulations impose requirements on employers to operate within stated exposure limits and to provide respiratory equipment where control limits are exceeded. As well, the regulations extend significantly further to rely on the IRS for the assessment of hazards and the development and implementation of control programmes, including the provision of appropriate medical surveillance. Thus, the Act applies in a special way to designated substances.

We shall consider this special relationship by focusing on the Regulation Respecting Asbestos. This Regulation contains eighteen sections and is currently supplemented by Codes for: Respiratory Equipment for Asbestos; Measuring Airborne Asbestos Fibres; and Medical Surveillance for Asbestos Exposed Workers.

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[18] Ibid., s. 8(12). The language of this section has proved problematic:

> . . . Arbitrator Barton in *Perley Hospital and Ontario Nurses Association* (1980), 29 L.A.C. (2d) 178 held that an employee was not entitled to pay when called in on her day off to attend a joint committee meeting. Barton held that this was not time from work and only "time so spent" was to be remunerated. The result can be defended in technical legal terms, for ss. 29(5) (accompaniment of an inspector) and 23(12) (inspection of a work refusal) specifically say that the time spent by a committee member in these situations is "deemed" to be work time. Section 8(12) does not use that magic word. [Morley Gunderson and Katherine Swinton, *Collective Bargaining and Asbestos Dangers at the Workplace*, Royal Commission on Asbestos Study Series, no. 1 (Toronto: Royal Commission on Asbestos, 1981), p. 8.15.]

(a) The IRS and Assessment

Section 6(1) of the Regulation Respecting Asbestos states that "Every employer to whom this Regulation applies shall cause *an assessment to be made in writing* of the exposure or likelihood of exposure of a worker to the inhalation or ingestion of asbestos." (Emphasis added.) Section 6(3) stipulates that the employer must consult with the joint health and safety committee in the preparation of this assessment and that the committee may make recommendations on same. Each member of the committee must receive a copy of the employer's assessment.[19] While the employer does not have to get the approval of the joint committee for the assessment, the joint committee, or a member thereof, has the right to notify a Ministry of Labour inspector in the event of any dispute between the employer and the committee regarding an assessment.[20] The inspector is then obliged to investigate the dispute and give a decision in writing.

(b) The IRS and the Control Programme

Section 7(1) of the Regulation stipulates that "Where the assessment discloses . . . that a worker is likely to inhale or ingest asbestos and that the health of the worker may be affected thereby," the employer must establish an asbestos control programme. This programme contains a number of elements which are set out in section 7(2), including engineering controls, work practices, hygiene practices and facilities, methods and procedures for monitoring record-keeping, medical examinations, and clinical tests.

In developing the control programme, the employer is again required to draw on the resources of the IRS by consulting with the joint health and safety committee. The committee may make recommendations regarding the control programme;[21] and the committee, or a member thereof, may notify a Ministry of Labour inspector in the event of a dispute with the employer regarding the asbestos control programme.[22] Once the control programme is in place, the employer is required to furnish each member of the joint health and safety committee with a copy of the programme and to acquaint every worker affected by it with its provisions, both in English and in the majority language of the workplace.[23]

(c) The IRS and Air Monitoring

The asbestos control programme must include provisions for "methods and procedures to monitor the concentrations of airborne asbestos in the work place and the exposure of a worker thereto."[24] The air monitoring procedures are set out in the Code for Measuring Airborne Asbestos Fibres. Air monitoring results must be conspicuously posted in the plant for fourteen days and furnished to the joint health and safety committee; they must be kept by the employer for a period of at least five years. It seems clear that the joint committee, as the institutionalized embodiment of the IRS, has a central role in ensuring that the ongoing results of air monitoring, critical to the achievement of the lowest practical exposure and to the enforcement of the control limits, are both known and reviewed.

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[19] Regulation Respecting Asbestos, s. 6(4).

[20] Ibid., s. 9(1).

[21] Ibid., s. 7(3).

[22] Ibid., s. 9(1).

[23] Ibid., ss. 10(1) and 10(2).

(d) The IRS and Medical Surveillance

The asbestos control programme must include provisions for medical examinations and clinical tests of asbestos-exposed workers.[25] These are paid for by the employer.[26] The employer must provide the physician who examines the worker with a copy of the records of the worker's exposure to airborne asbestos.[27] These records, together with those of the medical examinations and clinical tests, must be kept by the examining physician in a secure place for a period of up to forty years.[28] Where the physician is no longer able or willing to keep the records, they must be forwarded to the Chief Physician of the Ministry of Labour's Occupational Health Medical Service or his designate.[29]

Here the IRS incorporates another participant, the physician. Whether company physician or independent practitioner (a subject to which we shall return later in this chapter), the Regulation requires the physician to accept his own individual responsibility in the system.

C. The Role of the Ministry of Labour

C.1 The Occupational Health and Safety Division

The *Occupational Health and Safety Act* vests the government's responsibility for the health and safety of workers in Ontario in the Ministry of Labour, more specifically, in the Ministry's Occupational Health and Safety Division. This Division was assembled in 1976 from elements of the Ministries of Health, Natural Resources, and Labour to consolidate all provincial activities in occupational health and safety. The Division is led by an Assistant Deputy Minister, an Executive Director, and six Branch Directors.

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[24] Ibid., s. 7(2)(b).
[25] Ibid., s. 7(2)(d).
[26] Ibid., s. 13(1).
[27] Ibid., s. 14(2).
[28] Ibid., s. 15(1).
[29] Ibid., s. 15(2).

The Division's total complement is reportedly 732.[30] The Division is asked to provide the policy direction for the province with respect to the identification of potential hazards and the establishment of legislative standards, and to meet the day-to-day needs of the province for support, inspection, and enforcement with respect to healthy and safe workplaces.

Three branches of the Division — the Industrial Health and Safety Branch, the Mining Health and Safety Branch, and the Construction Health and Safety Branch — are primarily involved in developing and delivering health and safety field services to workers and employers. These services include ". . . inspection of work premises and practices for compliance with the health and safety legislation; advising and counselling employers, workers and suppliers of materials and equipment on the requirements of the legislation and more generally on health and safety concerns; enforcing the legislation; maintaining descriptive records of Branch activities and the problems dealt with; and contributing to the development of policy and legislation."[31]

The Industrial Health and Safety Branch is reported as employing 184 people, including 167 field staff. The Branch is decentralized into five administrative areas with twenty regions.[32] It has responsibility for all manufacturing, service, trade, transportation, storage, communication, and whatever other work sites are not covered by the Construction or Mining Branches or by federal health and safety legislation. In fact, approximately 150,000 industrial establishments in Ontario come under the jurisdiction of the Industrial Health and Safety Branch. In the period April 1, 1982 to March 31, 1983, the Branch made 46,480 inspections,[33] issued 56,435 orders, reviewed 1,962 plant submissions, investigated 118 refusals to work, and prosecuted 73 cases.[34]

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[30] Information in this section is drawn largely from Ontario, Ministry of Labour, "Annual Report 1982–1983," Draft. Statistical information is given for the period April 1, 1982 to March 31, 1983.

[31] Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, pp. 27, 31.

[32] Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Evidence of Mr. Walter Melinyshyn, 22 June 1982, Volume no. 43(A), p. 6.

[33] Each industrial establishment is on a regular inspection cycle ranging from once every three months to once every thirty-six months, depending on its record of accidents and fatalities, the number and nature of orders issued during inspections, labour-management relations, work refusals, etc. Inspection frequency can be increased or decreased on the advice of an inspector and approval of the regional manager. In addition to the above-noted cyclical reviews, known as Code 1 Internal Responsibility System inspections, inspectors can visit plants for other reasons, such as to check on the follow-up procedures to orders issued (Code 3); to make special calls, for example, in the event of a work refusal (Code 5); or to investigate accidents (Code 7).

[34] See also, RCA Transcript, Evidence of Mr. Walter Melinyshyn, 22 June 1982, Volume no. 43(A), pp. 5–15.

The Construction Health and Safety Branch is reported as employing 117 people, including 103 inspectors, with concerns for construction, renovation, and demolition activities of all types of structures such as buildings, tunnels, highways, and sewers. In fiscal 1982–1983, this Branch made 60,993 inspections, issued 24,378 orders, investigated 5 refusals to work, and prosecuted 245 cases.

The Mining Health and Safety Branch is reported as employing 94 people, including 27 inspectors, with concerns for some 700 mines, metallurgical plants, clay, shale, and peat workings, diamond drilling operations, and quarries, plus approximately 5,500 sand and gravel pits. In fiscal 1982–1983, this Branch carried out 5,673 inspections, issued 4,712 orders, investigated 13 refusals to work, and prosecuted 6 cases.

The field inspectors of the above three branches have a front-line role in ensuring compliance with the Act and the regulations made thereunder. They exercise their authority to enforce compliance by issuing orders pursuant to specific statutory and regulatory provisions.

An important part of an inspector's skills involves knowing when he needs to call on special assistance from the Division. In matters relating to occupational health, the inspector can draw on the support and expertise of the Occupational Health Branch of the Division. This is a staff branch of 187 people, most of whom are technical and professional personnel. The Branch includes the Occupational Health Medical Service, Occupational Health Hygiene Service, and Occupational Health Laboratory. The professional positions are filled by physicians, nurses, radiologists, occupational hygienists, engineers, toxicologists, scientists, and technicians. Inspectors can draw on any of the resources within this Branch simply by so requesting. For example, a physician may be asked to make a field visit to a plant and report to the Industrial Health and Safety Branch regarding the status of its medical surveillance programme. An engineer may be asked to look at problematic ventilation in a plant. In cases where the inspector requests an assessment of dust conditions in the plant, Occupational Health Branch hygienists will take samples and prepare an exposure assessment report.

The Occupational Health and Safety Division is rounded out by two other staff branches. The Special Studies and Services Branch has a complement of 68 people, including 15 involved in the Health Studies Service. This Branch is project-oriented, with assignments that focus on health and safety problems in specific industries or groups of workers, such as Dr. Murray M. Finkelstein's studies of mortality and morbidity among asbestos workers.[35] The Standards and Programs Branch has a complement of 73 people, including more than 40 professional staff. "This Branch is responsible for the development, co-ordination and evaluation of . . . the [Division's] policies and programs."[36] In relation to asbestos, this work has included the development of the Regulation Respecting Asbestos, and the development of training programmes and manuals for the field force.

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[35] See, for example, Murray M. Finkelstein, "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," *British Journal of Industrial Medicine* 40 (1983): 138–144.

C.2 The Ministry of Labour and the Internal Responsibility System

The Ministry, through its Occupational Health and Safety Division, deploys over 700 people in the goal of worker health and safety. This number is small when ranged against the estimated 170,000 workplaces in the province, many of them operating 7 days a week, 24 hours a day. Some plants are so large and diverse that detailed scrutiny of their operations would consume many days of inspector time.

In this setting, advocacy of the IRS is viewed by the Ministry as an essential tool of compliance. Even prior to the proclamation of the Act, Ministry inspectors were spending a good part of their time trying to convince parties at the workplace to take responsibility for health and safety. When the Act was proclaimed, more and more of the Ministry's activity focused on institutionalizing the IRS in Ontario industry.

Initially, Ministry inspectors spent a good deal of time helping companies set up joint committees and explaining their functions and responsibilities. Before the IRS was institutionalized, an inspector would arrive unannounced at a workplace, advise the plant manager or another senior officer of his intentions, begin an inspection, issue orders as necessary in a meeting with management, and return to see that the orders were complied with. Now, the routine has changed. The inspector still arrives unannounced, but now he must contact both the employer and the worker representatives of the joint committee and interview them to determine if there are any outstanding health and safety concerns so that these can become focal points of his inspection. The representatives accompany the inspector on the tour of the plant.[37] It is hoped that workers can see their representative(s) raising concerns, answering questions, and otherwise assisting the inspector to do his job. Then, in the words of a Ministry official, "The final part of an inspection consists of a discussion of the concerns, suggestions, and orders issued with worker and employer representatives, and a review of the existence and functioning of a joint health and safety committee."[38] During this discussion, the inspector will review the minutes of the last health and safety committee meeting and, if some committee recommendations have not been dealt with, he will try to ascertain the cause. "Where a specific recommendation is made that the employer does not act upon, and the recommendation concerns a breach or contravention of the Act or a regulation, a Ministry inspector, on being notified, can investigate and may issue an order where there are contraventions of the Act or regulations."[39] The inspector also, of course, raises his own concerns with the committee and gives a copy of his inspection report to the committee.

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[36] Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, pp. 35-36.

[37] *Occupational Health and Safety Act*, s. 28(3).

The inspection report includes orders issued to the company for contraventions of particular sections of the Act or of the regulations. The company is expected to fill out a Ministry of Labour "Order Follow-up" form indicating status of compliance with outstanding orders. The Ministry of Labour has recently made some administrative reforms in this area with respect to industrial establishments. Whereas the "Order Follow-up" forms used to be mailed to the employer thirty days after the inspection for completion regarding the status of those orders not yet complied with, these forms are now handed to the employer by the inspector before he leaves the premises. Moreover, whereas these forms used to be signed only by management upon completion, the Ministry is now encouraging the practice of securing the co-signature of a worker representative of the joint committee.[40] The latter is in response to concerns expressed by labour that "Order Follow-up" forms sometimes erroneously or prematurely report completion of orders.

The change in the inspector's routine and the responsibility placed on the joint committee to evaluate and make recommendations on conditions in the workplace, and to monitor compliance with orders, are intended to support the IRS and in turn to reinforce occupational health and safety.

The Ministry of Labour has also changed the way in which its technical staffs operate in relation to the IRS. In testimony before this Commission, a Ministry official noted that, before the Act, the Ministry would routinely monitor the air in industrial establishments where certain substances were known to be used. Since the passage of the Act, the establishment of joint committees in those sites that the Ministry had been monitoring, and the initiation of control programmes under the Regulation Respecting Asbestos, the Ministry has slowly changed its system to one where the company takes primary responsibility for air sampling and monitoring, with the Ministry performing an auditing role.[41] Likewise, the roles of the employer and of the joint committees in devising aspects of the control programme which deal with work and hygiene practices and with medical examinations have resulted in a devolution of the primacy once held by the Ministry of Labour, and hence represent a clear transfer of duties to the workplace in these areas.

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[38] RCA Transcript, Evidence of Mr. Walter Melinyshyn, 22 June 1982, Volume no. 43(A), p. 9.

[39] Ontario, Ministry of Labour, Occupational Health and Safety Division, *A Guide for Joint Health and Safety Committees and Representatives in the Workplace* (Toronto: Ontario Ministry of Labour, 1983), pp. 19–20.

[40] Ontario, Ministry of Labour, Memorandum from Ms. Pat C. Coursey, Staff Administrator, Industrial Health and Safety Branch to All Administrators and Operational Improvements Committee, Industrial Health and Safety Branch and Mr. J. Waterman, Standards and Programs Branch, 17 May 1983, pp. 10–11.

Beyond its activities directly in support of the IRS, the Ministry is responsible, in the last analysis, for enforcing the Act and regulations. Some of the complaints about the Ministry deal with the emphasis that the Ministry places on supporting the IRS at the expense of initiating prosecutions for violations of the legislation. Nevertheless, the Ministry does lay charges to enforce compliance and to deal with parties whose offences may have contributed to serious injury or fatalities.

We have received data which indicate that between April 1, 1980 and October 31, 1982, the Ministry of Labour launched 817 prosecutions and secured convictions in 666 of them, thus yielding an average success rate of 81.5%. The average fine levied against companies in successful prosecutions in that period was about $2,000. However, a telling instance of the extent to which the courts are beginning to take a severe view of violations came in December of 1982 when the Ontario Court of Appeal upheld a fine of

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[41] RCA Transcript, Evidence of Mr. Gyan S. Rajhans, 21 June 1982, Volume no. 42(A), pp. 17, 71-72.

$12,000. The Ministry has stated that "Since this decision, trends indicate higher fines on first conviction."[42]

From the prosecutions data we have reviewed, it is apparent that most of the charges have been in the area of safety rather than health. This may or may not be explained by the recency of the designated substances regulations; the effective enforcement of such regulations is a matter of major concern which we address below.

D. The Internal Responsibility System: Problems and Prescriptions

D.1 The IRS as the Foundation of Occupational Health and Safety

Our specific concern is, of course, asbestos and in particular the protection of workers from any adverse effects resulting from exposure to this substance. If we are concerned about the Internal Responsibility System generally, this is because it provides the foundation for Ontario's strategy to protect workers from asbestos exposure. Numerous complaints brought forward at our hearings indicate that this foundation is not yet regarded as secure.

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[42] Ontario, Ministry of Labour, "Current Issue Memorandum," 12 April 1983. See also, *Regina v. Cotton Felts Limited* (1982), 2 C.C.C. (3d) 287. While the upholding of a $12,000 fine has had the salutary effect indicated by the Ministry, the language used by the Ontario Court of Appeal is also instructive regarding the seriousness of breaching the *Occupational Health and Safety Act*. As Blair, J.A., speaking for the court, stated in his reasons at 293-294:

> Since, as far as we are aware, this is the first appeal against sentence under the Act to reach this court, it is incumbent on us to consider the proper principles governing the imposition of fines for this type of offence. As my brothers and myself made clear during argument, the range of fines imposed by the county court appears inordinately low for these offences . . . .
>
> The *Occupational Health and Safety Act* is part of a large family of statutes creating what are known as public welfare offences. The Act has a proud place in this group of statutes because its progenitors, the Factory Acts, were among the first modern public welfare statutes designed to establish standards of health and safety in the work place. Examples of this type of statute are legion and cover all facets of life ranging from safety and consumer protection to ecological conservation. In our complex interdependent modern society such regulatory statutes are accepted as essential in the public interest. They ensure standards of conduct, performance and reliability by various economic groups and make life tolerable for all. To a very large extent the enforcement of such statutes is achieved by fines imposed on offending corporations. The amount of the fine will be determined by a complex of considerations, including the size of the company involved, the scope of the economic activity in issue, the extent of actual and potential harm to the public, and the maximum penalty prescribed by statute. Above all, the amount of the fine will be determined by the need to enforce regulatory standards by deterrence . . . .

Organized labour has been especially critical of the IRS and made its views clear to us in its written and oral submissions. Labour feels that the concept is deceptive in that it has an appearance of protecting workers, while in practice it provides both management and the government with an excuse for doing as little as possible. Specifically, labour is critical that the committees may appear to give workers an involvement in health and safety while denying them the power actually to accomplish anything. Labour is also concerned that the Ministry uses the IRS to avoid the necessity of action. Labour claims that Ministry officials respond to problems in the workplace by telling the two sides — management and labour — to work difficulties out together. Labour claims that the powerlessness of workers in a "work it out yourselves" situation results in what is, in effect, collusive inaction between management and the government.[43]

The most important institutional arrangement in the IRS is the joint health and safety committee. The joint committee is the specific forum that brings management and labour together. The long-term success of the IRS depends on the successful operation of the joint committee. Spokesmen involved with such committees report the need for patience, commitment, and time. Even in an organization like Ontario Hydro where the commitment to worker protection extends from the Board of Directors to the workplace and where joint committees were in operation prior to the proclamation of the Act, the joint committees do not always work well. In fact, Ontario Hydro reported that a survey of joint committee members found that only 4 of 30 management and labour representatives felt that their committees worked "very well."[44] In other workplaces the situation may be less successful.

The study prepared by Professor Morley Gunderson and Professor Katherine Swinton for this Commission provided a review of the operation of joint health and safety committees:

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[43] See, for example, RCA Transcript, Submission by Mr. Robert Stewart on behalf of the Energy and Chemical Workers Union, 16 February 1981, Volume no. 1, p. 76; RCA Transcript, Submission by the Canadian Union of Public Employees, Local 27; and the Windsor Occupational Safety and Health Council, 27 March 1981, Volume no. 6, pp. 12-25; and RCA Transcript, Submission by Ms. Linda Jolley on behalf of the Ontario Federation of Labour, 28 January 1983, Volume no. 58, pp. 59-60. See also, *Canadian Occupational Health and Safety News* 6:2 (17 January 1983), p. 3; Cameron Wright, LeeAnne Mullis, and Lori Maynes, *Peterborough Toxics Profile* (Peterborough: Ontario Public Interest Research Group, Trent University, 1982); Elie Martel, "Not Yet Healthy, Not Yet Safe," a speech in the Ontario Legislature by Elie Martel, MPP, Chairman, Ontario New Democratic Task Force on Occupational Health and Safety, 26 April 1983; and Letter from Ms. Linda Jolley on behalf of the Ontario Federation of Labour to the Royal Commission on Asbestos, 31 January 1983.

[44] Ontario, Royal Commission on Asbestos, Exhibit II-56, Tab 8 [hereafter RCA Exhibit], in RCA Transcript, Evidence of Mr. Robert Wilson, 15 June 1982, Volume no. 40(B), p. 14: Presentation to the Royal Commission on Asbestos on the Internal Responsibility System in Ontario Hydro and Experience with Asbestos, Toronto, 15 June 1982, p. 10 and Table 2.

> An assessment of the efficacy of the joint health and safety committees is difficult to make, as the experience has been limited and empirical evidence of their operations is virtually non-existent. Some cautious generalizations may be attempted, but with the caveat that experience will vary in the 25,000 committees under the legislation. . . . Factors affecting operation will include the age of the committee, frequency of turnover of representatives, the financial position of the company, the general industrial relations climate, and the types of hazards in the workplace.[45]

The effectiveness of joint health and safety committees may vary depending upon whether the workers are organized or unorganized. Gunderson and Swinton have suggested that without the support provided by a labour organization, workers may find it very difficult to take advantage of the rights given to them under the Act and to exercise their responsibilities as members of a health and safety committee.[46] Further, where workers are not organized in unions, they are likely to be either uninformed of their rights or reluctant to use them fully. Ministry of Labour officials corroborated during our hearings that special efforts are required to make unorganized workers more effective participants in the internal responsibility approach to compliance.[47] In particular, the concept of the worker as an auditor of the workplace is less compelling where the individual does not feel protected by a union.[48]

The matter of labour organization aside, other circumstances can affect the degree of worker participation. In their study for this Commission, Professor Sally Luce and Professor Gene Swimmer pointed out that the operation of joint committees may be less effective where English is not the predominant language in the workplace, or where a variety of languages are spoken, thus impeding communication among workers and between workers and their representatives on the health and safety committee. In addition, worker involvement may be constrained where workers are culturally reluctant to "get involved," or where workers are transient and relatively unskilled.[49]

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[45] Gunderson and Swinton, *Collective Bargaining and Asbestos Dangers at the Workplace*, p. 8.10.

[46] Ibid., p. 8.21.

[47] RCA Transcript, Evidence of Mr. Walter Melinyshyn, 22 June 1982, Volume no. 43(A), pp. 50, 54.

[48] RCA Transcript, Evidence of Mr. Arthur L. Gladstone, 30 June 1982, Volume no. 47(A), pp. 105–106.

[49] Sally Luce and Gene Swimmer, *Worker Attitudes About Health and Safety in Three Asbestos Brake Manufacturing Plants*, Royal Commission on Asbestos Study Series, no. 6 (Toronto: Royal Commission on Asbestos, 1982), pp. 6.2–6.3.

In addition to problems of participation by workers, there are clearly problems at the management level. Management's concern about health and safety is variable among workplaces. Some are highly concerned; some less so. Where management is less concerned, the seed of internal responsibility will have more difficulty germinating. As well, management's propensity to consult workers can be highly variable.

The problems we have referred to above affect all aspects of the operation of joint health and safety committees, not only their effectiveness in dealing with designated substances. Solutions to these problems should be designed with a complete view of the full range of responsibilities assigned to these committees. The details of such solutions lie well beyond our terms of reference. Inasmuch as the general effectiveness of the Internal Responsibility System is the all-important foundation of measures intended to protect workers from hazards of asbestos, we urge the Ministry of Labour to undertake a systematic review of the operation of the joint health and safety committees. We note that the Advisory Council on Occupational Health and Occupational Safety has struck a task force on the *Occupational Health and Safety Act*, including the operation of the joint committees. We further note that the recent Task Force on Occupational Health and Safety of the Ontario New Democratic Party, which catalogued a number of serious problems, concluded among other things ". . . that the Internal Responsibility System as it now exists only works when the company and the union are equally committed to making it work."[50] Similar views were expressed at our hearings and lead us to emphasize the importance, in any Ministry review of joint committees, of seeking to identify instances in which joint committees have operated successfully, the factors that account for this success, and the extent to which the labour and management participants in successful operations can contribute to the dissemination of information to other workplaces by acquiring a role in training programmes or by other means. In line with these considerations, we recommend that:

8.1 The Ministry of Labour should undertake to identify instances of the successful operation of joint health and safety committees and the means whereby the factors that account for this success can be disseminated through information and training programmes, drawing upon the review by the Advisory Council on Occupational Health and Occupational Safety and such other analyses as are necessary.

D.2 The IRS and Training

Training has long been recognized as a key adjunct of occupational health and safety, particularly in the realm of accident prevention. The new and welcome emphasis on health augments the need for training. In addition, the premium that the IRS places on joint labour-management involvement in the health and safety of the workplace invites joint rather than separate training programmes. In this regard, current training efforts in Ontario strike us as lagging behind the basic philosophy of internal responsibility.

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[50] Martel, "Not Yet Healthy, Not Yet Safe," p. 8.

Training programmes are available relating to worker health and safety through the Accident Prevention Associations, which are funded by the Workers' Compensation Board. It has been submitted to us, however, that labour does not place its full confidence in the Accident Prevention Associations, viewing them as being biased towards management.[51] There is thus some suspicion of training programmes offered through these associations. Meantime, the Ontario Federation of Labour (OFL) has developed training programmes of its own, which have received substantial usage. The OFL has informed us of more than a dozen workplaces where OFL training for worker health and safety has been used, including General Motors of Canada Limited, Northern Telecom, American Can Corporation, and Algoma Steel. These training programmes were offered in many cases to management as well as to labour. The OFL training programmes are supported in part by the Ministry of Labour, through its apportionment from the profits of the Ontario Lottery Corporation.

It may be that fundamental reform of the Accident Prevention Associations is required if labour is to have full confidence in these associations. We understand that the Advisory Council on Occupational Health and Occupational Safety has recommended to the Minister of Labour that representation on the Council of Safety Associations be altered to achieve a balance between labour and management, and that the Council of Safety Associations be encouraged to enter into appropriate arrangements with labour and the Accident Prevention Associations to develop occupational health and safety training programmes jointly run by management and labour.[52] We view the organization of the safety associations as a matter that lies beyond our terms of reference, and note that Professor Paul C. Weiler will be addressing it in a future report to the Minister of Labour on workers' compensation.[53]

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[51] RCA Transcript, Submission by Mr. Ed Hunt on behalf of the United Electrical, Radio and Machine Workers of America, 20 February 1981, Volume no. 5, p. 86.

[52] Ontario, Ministry of Labour, Advisory Council on Occupational Health and Occupational Safety, "Advisory Memorandum 81-II on Prevention in Occupational Health and Safety Through the Workmen's Compensation Board and its Accident Prevention Associations," in *Fourth Annual Report, April 1, 1981 to March 31, 1982* (Toronto: ACOHOS, 1982), pp. 57-70, especially Recommendations 81-6 and 81-7, p. 67.

[53] Paul C. Weiler, *Protecting the Worker from Disability: Challenges for the Eighties*, a report submitted to Russell H. Ramsay, Minister of Labour ([Toronto: Ontario Ministry of Labour], April 1983), p. 5

Nevertheless, we do believe that a training structure more in keeping with the joint philosophy of the IRS might result from giving further encouragement to management and labour to design their own co-operative training programmes when they feel this would better serve the needs of their workers than programmes currently offered by the Accident Prevention Associations. In this connection, we note that the *Workers' Compensation Act* applies levies on employers which do not take account of the cost of training programmes that have been mounted outside the framework of the associations in co-operation with labour. In order to encourage the development of joint programmes that are in line with the philosophy of the IRS, we consider it highly desirable that employers who choose to organize training courses in full concert with labour unions should enjoy a reduced assessment. This simple step would likely multiply the number of joint training programmes. Accordingly, we recommend that:

8.2 The Workers' Compensation Act should be amended to provide that where an employer establishes or contributes from his own resources to the financing of a joint labour-management programme for the purpose of education in accident or disease prevention, and the Board is satisfied that the programme is serving the same purpose as an Accident Prevention Association programme, the Board may reduce the assessment for which the employer is liable as a member of a class represented by an Association.

Whatever the auspices under which training programmes are offered, it is critically important that members of joint committees take advantage of the training opportunities that are available. The Ontario Federation of Labour made a particularly strong point about the training that is necessary if worker representatives on joint committees are to be equipped to deal with health as distinct from safety issues. In the absence of training, workers may be quite unable to carry on effective dialogue with technically qualified management representatives on such health issues as the identification of hazardous substances, the particular hazards they pose, the need for improved measures of control, and the practicality of alternative control measures. Furthermore, workers coming directly off the shop floor may be unfamiliar with the skills and techniques of effective committee work. For both the substance of health issues and effective participation in joint committees, labour representatives in particular need to take advantage of the training opportunities that are available.

This raises the question of whether labour representatives on joint committees should receive pay while attending training courses. Under section 8(12) of the *Occupational Health and Safety Act*, a committee member is only paid for such time from his work as is necessary to attend committee meetings, conduct a periodic inspection of the workplace, or investigate a serious accident. We consider it beyond our terms of reference to address the matter of paid time for health and safety training in general, but well within our mandate to consider the issue of paid time for training with an occupational health content in workplaces where a designated substance such as asbestos is present. We accept the validity of the contention of the OFL that occupational health training is particularly necessary if labour representatives are to participate effectively in the work of joint committees. The presence in the workplace of a substance which, like asbestos, has been deemed sufficiently hazardous to warrant designation in our view justifies paid time for worker training. Given an enabling clause in the legislation, the place in which to make this stipulation is the appropriate designated substance regulation. Accordingly, we recommend that:

8.3 ***The Occupational Health and Safety Act should be amended to provide that, where stipulated by regulation, members of a joint health and safety committee may be entitled to paid time from work for the purpose of occupational health and safety training; the Regulation Respecting Asbestos should be amended to stipulate that members of joint health and safety committees shall be entitled to paid time from work for the purpose of occupational health training in programmes approved by the Minister.***

D.3 The Importance of Informed Workers

The Act places considerable onus on workers to serve as their own health and safety inspectors to ensure compliance with legal requirements and more particularly the absence of any situation likely to endanger their health and safety in the workplace. The Regulation Respecting Asbestos attempts to equip workers to exercise this responsibility by requiring that employers ". . . acquaint every worker affected by the asbestos control program with its provisions."[54] This provision is intended to ensure that workers are aware of, for example, the importance of ventilation equipment, dust covers, and respirators and should, in keeping with the Act, allow the worker to avoid situations likely to endanger his health. But the worker would be even better equipped to avoid danger if he were aware of the nature of the hazard.

This matter is covered in part by the Code for Medical Surveillance which accompanies the Regulation. Section 5 of the Code, entitled Health Education, prescribes that at the pre-placement and periodic medical examinations all workers are to be ". . . advised of the hazard from asbestos and smoking. . . ." Nowhere, however, does the Regulation stipulate that workers are to be informed about the starkest fact concerning asbestos-related disease: the existence of a dose-response relationship. We attach the greatest importance to ensuring that every asbestos worker should know that the lower the exposure to asbestos, the lower the risk of disease.

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[54] Regulation Respecting Asbestos, s. 10(1).

As we stated at the beginning of this chapter, what we regard as fundamental to the matter of control limits is the duty which the Regulation imposes on every employer to take all necessary measures to ensure that exposure to asbestos "is reduced to the lowest practical level." This means that the control limits are not norms but maxima; the employer's obligation is to conform to what is commonly called the ALARA principle (As Low As Reasonably Achievable). We appreciate the point made at our hearings by counsel for the Asbestos Victims of Ontario and the Iron Workers' Union, Mr. David Starkman, that it would be very difficult for the Ministry to prosecute an employer for failing to achieve the lowest practical level if in fact all measurements met the numerical control limit.[55] It follows that if the ALARA principle is to have any operational significance, workers must be aware that it is part of the law, so that they can, through their joint health and safety committees, insist on the performance the Regulation requires. It is therefore crucial that workers be made aware of this provision, which we believe has regrettably drawn far less attention than the numerical control limits. The Act and regulations focus communications on the joint committee rather than on rank and file workers. Thus, it is generally left to labour and management, as part of the IRS, to inform workers of their rights and responsibilities.

In this setting, we conclude that it is imperative that the Regulation Respecting Asbestos stipulate that an essential element of every control programme should be to inform workers of both the existence of a dose-response relationship and of the fact that the Regulation establishes the duty of the employer to ensure that exposure to asbestos is reduced to the lowest practical level. We therefore recommend that:

8.4 The Regulation Respecting Asbestos should be amended so as to require that every asbestos worker shall be informed of:

- ***(i) the existence of a dose-response relationship for asbestos-related disease, namely, the lower the exposure, the lower the risk of disease; and***
- ***(ii) the fact that the Regulation establishes control limits as maxima and imposes on the employer the duty to take all necessary measures and procedures to ensure that the time-weighted average exposure of a worker to airborne asbestos is reduced to the lowest practical level.***

In the ongoing reality of the workplace, the operational effectiveness of the ALARA principle will hinge in large part on the capacity of employers and workers to observe the behaviour of measured exposure levels over time. Are these levels steady, declining, or drifting upwards? To answer this question requires that it be possible to compare dust levels at any particular time with previously existing dust levels. This requirement has implications for deciding what air sampling to undertake, how to summarize the results of that sampling, and how to present the results.

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[55] RCA Transcript, Submission by Mr. David Starkman on behalf of the Asbestos Victims of Ontario and the International Association of Bridge, Structural and Ornamental Iron Workers, Local 721, 28 January 1983, Volume no. 58, p. 17.

We have analyzed the hygiene data taken by the Ministry in an Ontario asbestos manufacturing plant over a period of several years. These data revealed considerable variations in dust levels from one work station to another, from one process in the plant to another, and from one measurement date to another. These considerable and significant variations mean that a single average dust level calculated for a plant at a particular time is not especially meaningful. The average would depend very much on where samples were taken, as well as on the other factors that cause variability in dust levels. If current readings are to be compared with past ones, this must be done separately for each area of the plant that has significantly different dust levels. Furthermore, a meaningful comparison can only be made when a number of samples are available, because of the high degree of variability from one sample to another. This requires that the several samples taken in a particular area of a plant be summarized, by calculating the average, median, or geometric mean of those samples, to yield a single statistic that may be compared with the corresponding statistic for previous periods. In Chapter 7 we present what appears to be the appropriate summary statistic,[56] but careful study might reveal that some other summary statistic would better represent highly variable data such as these.

Once such a summary can be presented for each relevant area of the plant for each air quality assessment in the plant, there is still the question of how successive summary statistics can be compared with each other. A simple comparison would be to maintain a plot or graph of these data over, for example, a one- or two-year period. This is probably desirable. Once again, however, the variability of the data means that any given assessment statistic may be strongly influenced by random variations. A more precise test of whether air quality for a given period is in fact worse or better than that of the recent past would be to compare a weighted average of the statistics from the last few periods, with declining weights attached to the early readings, to the average for the previous year or two. Such a comparison would avoid generating alarm when random events cause the readings to increase, while simultaneously signalling persistent upward trends in the data. We believe that information such as that just discussed should be prepared for each asbestos-using plant on a regular basis, and posted for a considerable period of time for the information of both workers and management. Accordingly, we recommend that:

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[56] See Chapter 7, Section B.4 and note 18 therein.

8.5 Section 12 of the Regulation Respecting Asbestos should be amended to require that each time monitoring of airborne asbestos takes place, an appropriate summary statistic of the resulting fibre concentrations be calculated for each area of the plant, where areas are defined as portions of the plant in which dust levels are reasonably similar. The results for each area to be posted under section 12 should include these summary statistics. In addition, a summary of the average dust level in each area over the past one or two years should be calculated and compared with the current dust levels and with a weighted average of recent dust levels. The results of the monitoring and of this analysis should be posted as specified in section 12(a), but for a period of at least two months, or until rendered obsolete by subsequent measurements, rather than for the fourteen-day period specified in the existing Regulation.

Another problem with the sampling aspect of a monitoring programme arises from the need to ensure that the equipment is properly calibrated and that the use of the equipment is such that the resulting fibre counts are fair and representative measures of the dust concentrations to which the worker is exposed. This requires a reasonable degree of competence and good intentions on the part of the individual responsible for conducting or supervising the air monitoring programme. It also requires that workers should feel assured that the proper procedures are in fact being followed. In line with the philosophy of the IRS, we shall first address the requirement of ensuring the competence of the individual responsible for supervising the air monitoring programme and then the importance of worker assurance.

The former is a straightforward matter of acceptable qualifications. Here the Regulation Respecting Asbestos presents a curious anomaly as between its Code for Medical Surveillance and its Code for Measuring Airborne Asbestos Fibres. Because the Medical Surveillance Code refers to the role of the examining physician, it automatically ensures that this role will be filled by an individual with accredited professional qualifications. The Measurement Code, on the other hand, contains instructions at a level of technicality such that they could only be understood by a trained individual, but makes no reference whatsoever to what kind of individual might possess the necessary qualifications. The Regulation therefore appears to assume that qualified individuals will be in charge of air monitoring, and presumably leaves detection of unsuitable work by unqualified individuals to the inspection process.

We consider this to be less than satisfactory. In respect of each plant with an asbestos control programme, there should be an individual with designated responsibility for designing representative air sampling procedures, for seeing that the Code for Measuring Airborne Asbestos Fibres is respected, and for ensuring that equipment is properly calibrated. This individual should be certified as competent by the Occupational Health and Safety Division of the Ministry of Labour and liable to the loss of his certificate if the findings of inspectors so warrant. We permit ourselves to hope that the initial issuance of certificates could be simply a matter of having the Occupational Health Hygiene Service endorse the qualifications which the individuals now in charge of asbestos air monitoring in plants, whether as employees or contracted professionals, already possess. If this hope proves unfounded, the rationale for our prescription is that much stronger. We therefore recommend that:

8.6 The Regulation Respecting Asbestos should be amended so as to require that in every plant with a control programme, there shall be an individual certified by the Occupational Health Hygiene Service who will be designated as responsible for designing representative air sampling procedures; for seeing that the Code for Measuring Airborne Asbestos Fibres is respected; and for ensuring that equipment is properly calibrated.

In the matter of worker assurance that air monitoring is being suitably implemented, we are mindful of the extent to which certain aspects of air sampling are sufficiently complex to baffle lay-persons. This, of course, argues in favour of the certification we have prescribed. On the other hand, there are aspects of air monitoring (notably, observing the proper calibration of equipment and the question of where to sample and when) for which lack of technical qualifications is not a barrier to worker participation.

We attach importance to a worker role at the level of observing calibration and suggesting what portions of a plant's operations should be included in a representative air sampling programme. With respect to the latter, a worker's first-hand knowledge of conditions on the plant floor can be particularly valuable. While all the worker members of a joint health and safety committee can be potentially of assistance, we look as a practical matter to a particular role for the worker representative who, pursuant to section 8(8) of the Act, must be designated to make periodic inspections of the workplace. It is desirable, as we point out in Chapter 7, to include worker representatives in the task of fibre counting. (See Recommendation 7.8.) Whether or not there is worker involvement in this task, the Regulation should require that the worker representative appointed pursuant to section 8(8) of the Act observe the calibration of air sampling equipment and be consulted with respect to what constitutes representative air sampling. On the basis of these activities, the worker representative will likely promote informed discussion at the level of the joint committee and be a source of reassurance to his fellow members on the committee. His role will in some ways parallel that of the worker representative in mines, concerning which *The Report of the Joint Federal-Provincial Inquiry Commission into Safety in Mines and Mining Plants in Ontario* (the Burkett Report) commented at some length. This is appropriate in that a designated substance workplace, like a mine, warrants special precautionary measures. In this regard, we note that the Burkett Report recommended that the worker representative in mines should be afforded preparation time, together with office and secretarial assistance, so as to enhance his effectiveness,[57] and register our considered opinion that the same should apply in workplaces in which asbestos is present. In line with these considerations, we recommend that:

8.7 The Regulation Respecting Asbestos should be amended to require that the worker representative appointed pursuant to section 8(8) of the Occupational Health and Safety Act:

- *(i) have the opportunity to observe, on his plant tour, the calibration of air monitoring equipment;*
- *(ii) be consulted on the adequacy of representative air sampling procedures; and*
- *(iii) be afforded preparation time, together with office and secretarial assistance, beyond that already recognized for the preparation of minutes of committee meetings.*

D.4 The IRS and the Role of Management

The critical importance which we attach to the ALARA principle that is embodied in the Regulation Respecting Asbestos leads us naturally to the role of management in this regard. Management attitudes towards the control programme will strongly influence the level of exposure below the control limits that will be considered practically achievable. More generally, these attitudes will undoubtedly have a considerable influence on the vigour with which management pursues health and safety programmes through the IRS and its degree of co-operation with the worker members of joint health and safety committees.

We believe that management attitudes towards worker health will be significantly conditioned by the extent to which supervisory personnel understand the risks that can be presented to workers by various situations. This calls for training designed to impart the requisite knowledge. It also calls for a setting in which supervisory personnel with the requisite training will be on hand whenever plant operation or maintenance entails worker exposure to asbestos. The Luce and Swimmer study uncovered allegations that some of the dustiest operations occurred on the night shift. In this light, we recommend that:

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[57] Canada/Ontario, *The Report of the Joint Federal-Provincial Inquiry Commission into Safety in Mines and Mining Plants in Ontario: Towards Safe Production* (Burkett Report), Kevin M. Burkett, Chairman, vol. 1 (Toronto: April 1981), p. 79.

8.8 The Regulation Respecting Asbestos should be amended so as to require that every employer with a control programme must select persons who have supervisory positions for training in the nature of the hazards posed by asbestos, and in identifying and dealing with the specific workplace situations that may be most hazardous. The number of persons trained should be sufficient to ensure that one such person will be on duty in the plant at all times when its operation or maintenance entails worker exposure to asbestos.

D.5 The IRS and the Role of Ministry Inspectors

The testimony of past and current officials of the Ministry of Labour underlines the Ministry's support of the IRS. Inspection routines, accident and complaint investigations, responses to "right to refuse" situations, and routine monitoring activities have in principle been conditioned by the need to support and encourage the IRS. Indeed, labour representatives argue that the Ministry has gone too far, replacing immediate action with exhortations of support for joint committee action. We shall address the Ministry's enforcement role later in this chapter; for the moment, we take the submission of labour representatives as further evidence of the Ministry's commitment, through its inspectorate, to the IRS.

The Ministry has chosen to organize its inspectorate along geographical lines rather than specializing by industry or type of hazard. What speaks in favour of this mode of organization is that the front-line inspectorate gives the Ministry a comprehensive window on all aspects of health and safety in the workplace and an opportunity to monitor and encourage the development of the IRS in all its several dimensions. But this mode of organization also means that the front-line inspectors will of necessity be generalists. There is an uncertain demarcation line beyond which the Ministry's expectations of its inspectors may be super-human. This led Professor G. Bruce Doern, Professor Michael Prince, and Mr. Garth McNaughton, in their study for this Commission, to suggest that the Occupational Health Branch might assume the front-line inspectional role for matters of occupational health.[58] As we shall explain below, we ourselves conclude that there is a need for a specialized inspection unit where designated substances are present in the workplace. But such a unit should not operate in lieu of the Ministry's generalist inspectors. The comprehensive window which these inspectors give to the Ministry on each workplace should not be discarded. In reaching this conclusion, we draw in part on a review, conducted at our request by our staff, of the Ministry files for the specific brake plants whose workers were surveyed by Luce and Swimmer. In that these files revealed a gratifying degree of inspector awareness of the problems independently identified by Luce and Swimmer, the value of the Ministry's comprehensive window is evident.

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[58] G. Bruce Doern, Michael Prince, and Garth McNaughton, *Living with Contradictions: Health and Safety Regulation and Implementation in Ontario*, Royal Commission on Asbestos Study Series, no. 5 (Toronto: Royal Commission on Asbestos, 1982), p. 5.14.

Of course, there is a need to ensure that generalist inspectors have a degree of training in matters of occupational health. If only by force of history, experienced front-line inspectors have been better equipped to deal with accident prevention than disease prevention. They must be sensitized to health hazards, and they must know what sources of expertise in the Occupational Health Branch are on tap to be called in. They require a basic understanding of control methods such as ventilation, a general grasp of the requirements of designated substances regulations — especially the ALARA principle — and an understanding of the principal elements of designated substance control programmes.

According to Ministry testimony before this Commission, no more than half of the current front-line inspectors have undergone such basic occupational health training.[59] We have not reviewed the adequacy of the content of this training. We urge the Ministry to do so, particularly with an eye on whether the health training of inspectors might be integrated with available joint labour-management training programmes. This would be much in line with the Ministry's policy of supporting the IRS. The Ministry should seek as expeditiously as possible to enhance the proportion of its inspectorate that has received health training. In the meantime, the Ministry should make every effort to ensure that the inspectors assigned to visit plants where designated substances are present are individuals who have received basic health training. The present asbestos-using industry consists of a small number of firms that are primary asbestos users — according to Chapter 6 of this Report, no more than a dozen. In addition to these, a few dozen more use non-trivial amounts of asbestos and are therefore covered by the Regulation Respecting Asbestos. If this pattern is typical of most of the industries in which designated substances are used, it may well be feasible to ensure that the inspectors assigned are those who have already received health training. In line with these considerations, we recommend that:

8.9 The Ministry of Labour should review the content of the basic occupational health training it offers to Industrial Health and Safety Branch inspectors with a view to its adequacy and to whether it might be integrated with available joint labour-management health training programmes, and the Ministry should seek as expeditiously as possible to enhance the proportion of its inspectorate that has received occupational health training. As an interim measure, the Ministry should make every effort to ensure that the inspectors assigned to

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[59] RCA Transcript, Evidence of Mr. Walter Melinyshyn, 22 June 1982, Volume no. 43(A), p. 31.

plants where designated substances are present are individuals who have received basic occupational health training.

E. Beyond the Internal Responsibility System

E.1 A Designated Substances Enforcement Unit

The IRS is at once the desirable and the practical approach to occupational health and safety. This is because the central goal of accident and disease prevention hinges on the concerted efforts of all who are associated with the workplace, and because no government agency could conceivably ensure that health and safety are fully policed. If there are problems with the IRS, this is due in part to its recency. It evidently stands in need of improvement. Given what we view as the greater complexity of disease prevention, we would expect the IRS to approach its full potential in occupational safety before it becomes as effective in occupational health. The recommendations we have made so far are intended to lend direct support to the favourable development of the IRS in matters of health; further recommendations made in Chapter 14 are likewise intended to lend direct support. We believe, however, that *indirectly,* the development of the IRS in the domain of occupational health can be abetted through a measured dose of firm policing. The litany of complaints we have heard about the problem of dealing with designated substances is such as to cause us grave concern if only because it bespeaks a lack of worker confidence in the occupational health regime. There is a perceived lack of response by the Ministry of Labour to complaints from joint health and safety committees and a perceived lack of prosecution in cases where orders have not been complied with within the specified time. While these problems may occur in a variety of health and safety areas, they are of special concern with respect to designated substances because it may be more difficult for joint health and safety committees to maintain pressure over a period of time where the harmful effect is not obvious and lies far in the future. We believe that the Ministry should effectively respond to this problem by creating a Designated Substances Enforcement Unit (DSEU).

The DSEU should be constituted so as to command the full range of multidisciplinary technical expertise necessary to enforce designated substances regulations. Its focus would be on health; its mandate, to enforce the regulations. The DSEU should be headed by a Ministry official with the rank of Branch Director. This official, among his other qualifications, should have thorough knowledge of the powers of the Ministry of Labour and of investigative procedures, including circumstances where search warrants should be sought and may be issued. This official would have the authority to launch special, unannounced inspections of plants in which designated substances are in use and to commandeer a team of appropriate Ministry experts assembled separately for each plant visit. The composition of the team would depend upon the designated substance in question and the types of expertise necessary to examine compliance with the regulations at the particular workplace. Thus, other than for its Director, the DSEU would not consist of additional full-time staff of the Ministry, but would rather be a term applied to regular staff assembled on particular occasions for a particular purpose. A visit from the DSEU would differ from an ordinary visit by a Ministry of Labour inspector in several respects. First, each visit would be made by a team consisting of a number of experts, rather than a single inspector. The team would presumably include one or more experts in control methods for the designated substance, which in the case of asbestos would require an individual expert in ventilation and in the use of respirators; an expert in measuring concentrations of the designated substance, who could both perform some measurements and carefully review the measurement records kept in the workplace to check their completeness and accuracy; and someone who would be competent to determine whether all of the regulations applying to designated substances were being fully complied with in the workplace. Where the size of the workplace was substantial, several experts of each type might be required in order carefully to examine the plant on a single visit. The DSEU might visit outside regular business hours, where a plant normally operated outside those hours.

In deciding whether to launch a visit, the Director of the DSEU could appropriately take account of the advice of inspectors, workers, unions, police, or any member of the public who suspects that regulations are not being met. He could also choose randomly from the list of workplaces where designated substances are used. In the case of asbestos, there are about a dozen major users in Ontario, and a few dozen more firms that from time to time use small quantities of asbestos. The Director of the DSEU would choose a strategy for visiting these plants in a way such as to maximize the likelihood of identifying and dealing with significant problems. The visits of the DSEU would be above and beyond the normal visits by Ministry inspectors and should have no impact on such visits, unless the DSEU recommended that a follow-up visit by an inspector be performed. Following an inspection, the DSEU would file a report with the Ministry, the employer, and the worker representatives on the health and safety committee, which included general findings at the workplace, compliance orders with specific expiry dates attached, stop work orders, if necessary, and recommendations for prosecution where significant violations were detected. The DSEU would pay special attention to compliance orders from field inspectors that had expired but were not complied with, and to long-standing complaints by the joint health and safety committee. Its attitude should reflect the importance of achieving compliance with control limits and the ALARA principle as a condition for using a designated substance in the workplace.

The philosophy of the DSEU would evidently differ from the general compliance philosophy of the Ministry of Labour. The DSEU is designed to provide a tough enforcement capability that is not generally present in the Ministry's reliance on the Internal Responsibility System. The DSEU might be less flexible and less generous in dealing with compliance problems than would be an inspector who is inevitably in a long-term relationship with the parties and a consultant-promoter of the IRS. Of course, the DSEU cannot be so rigid and uncompromising that it completely undermines the Ministry's attempts to develop a co-operative attitude between the employer and the Ministry and between the employer and the employee. It is not our intention through implementation of the DSEU to undermine or weaken the IRS. Rather, we intend the DSEU to lend the IRS indirect support.

Inspection by the DSEU could be of assistance to employers who may be complying with standards but may be unable to convince employees of their performance. It should alter the perception of labour groups and workers who feel that current inspections lack substance and that the Ministry is too lenient. It should help the Ministry to ensure that its statutes are enforced without turning its entire Division into a police force. The multidisciplinary DSEU team will likely find areas for improvement in the workplace even where the regulations are being met. The report of their findings will provide opportunity for the joint committee to focus on specifics.

In several respects the DSEU as described addresses the problems of enforcement that have been brought to our attention. First, the multidisciplinary nature of each inspection team recognizes the range of specialties involved in designated substance control while simultaneously allowing for, and indeed abetting, the continued and desirable role of generalist inspectors deployed on a geographical basis. Second, the DSEU recognizes that the need for a positive Ministry enforcement role may be greater in matters of health than in those of safety. Workers and first-line supervisors may be the most important parties in safety; in health, the onus shifts to the engineer, technician, and scientist. Third, the submissions from labour and the study by Luce and Swimmer have indicated that there is a good deal of suspicion in the workplace that conditions respecting occupational health are not as they should be. The routine inspection from the Ministry is sometimes not reassuring and indeed may not even be noticed. A blitz from a well-equipped Ministry team would be far more impressive and could improve worker perception. Fourth, follow-up to the kinds of allegations brought to the attention of this Commission involves police work as much as inspection. Rumours that a plant operates its dirty machines only in the middle of the night or that a plant with non-English speaking immigrant workers is ignoring the law may require the kind of follow-up that is more typical of police work than routine field inspection. In a different but related context, the Ontario Ministry of the Environment found this to be the case and has established a special police enforcement unit.[60] Finally, we have become aware that a problem with the Ministry of Labour lies in appearance: it just does not look like a tough enforcement organization. Appearances are important, not only in satisfying workers but also in convincing employers and managers that control actions are essential. Accordingly, we recommend that:

8.10 The Ministry of Labour should create a Designated Substances Enforcement Unit (DSEU), headed by an official with the rank of Branch Director. This official would have the authority to launch special, unannounced inspection visits of plants in which designated substances are in use and to commandeer a multidisciplinary team of appropriate Ministry experts assembled separately for each inspection visit. Each DSEU team should have all the powers of a Ministry of Labour inspector.

E.2 Medical Surveillance: The Examining Physician

The Regulation Respecting Asbestos requires in section 13(1) that the worker, at the expense of the employer, shall undergo medical examination and clinical tests. Sections 14, 15, and 16, as well as the Code for Medical Surveillance, cover the information to be gathered under the medical surveillance programme and the maintenance of the records kept therefor. It is not clear from the Regulation, however, who selects the physician to conduct the examination. Labour has indicated its preference that the worker be entitled to choose the examining physician. One document filed with the Commission, a memorandum from the Director of the Legal Services Branch of the Ministry of Labour concerning selection of the physician for conducting examinations regarding lead, stated that under the Regulation the employer would select the physician to conduct this examination.[61] The Ministry of Labour is developing a general guide to the designated substances regulations which will address the issue. We have been informed that the Ministry believes that the physician should be selected in consultation with the joint health and safety committee. However, the Ministry recognizes that there is no requirement in law for a single physician to conduct the examinations in the plant, and that individual workers can choose their own family doctors.[62]

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[60] The Ministry of the Environment, given problems of non-compliance with regulations, created a set of special enforcement units (SIUs) to deal with these problems. Announced by the Minister in October 1980, the SIUs consist of one or two staff at each regional office who have been trained in investigative methods at police college. The SIUs are apparently highly effective in gathering the background information necessary to support the successful prosecution of a serious offender.

[61] RCA Exhibit II-60(c), in RCA Transcript, Evidence of Mr. Walter Melinyshyn, 22 June 1982, Volume no. 43(A): Ontario, Ministry of Labour, Memorandum from Mr. Paul Hess, Director, Legal Services Branch to Mr. Alan Heath, Director, Standards and Programs Branch, 22 April 1982.

We received a number of representations on the issue of who is an appropriate examining physician. Workers are frequently mistrustful of "company doctors," believing them not to be completely independent in judgements made about worker health. On the side of company physicians, concerns have been expressed to this Commission that physicians selected by individual workers might not necessarily have the competence necessary for evaluating specific work-related diseases which are often not common in the general population. It has become apparent to us that the degree of controversy over who should be the examining physician has been heightened by the role which the Regulation assigns to this individual. In this regard, section 16(1) of the Regulation gives the examining physician the authority to declare whether the employee is ". . . fit . . . fit with limitations or unfit, to work in an asbestos exposure." In testimony before us and in subsequent correspondence with this Commission, Dr. Jerome J. Vingilis, formerly of the Ministry of Labour, expressed his concern that certain company physicians might abuse the authority to declare a worker unfit.[63]

Later in this Report, for reasons stated in Chapter 14, we recommend that the examining physician should cease to have the authority to declare workers fit or unfit to work in an asbestos exposure. This removes one source of the controversy over who the examining physician should be. In that the controversy remains, we express the strong view that, ideally, the examining physician should be chosen jointly by labour and management. More generally, we believe that the most important factor in physician selection should be the competence of the physician who is to examine the worker.

We consider that the most likely and convenient source of expertise is to be found among company doctors. Such physicians, by the inherent nature of the positions they hold, are at once repositories of experience in occupational medicine and, in the specific domain of asbestos, bound to become familiar with the provisions of the Code for Medical Surveillance. The interests of employers, employees, and company physicians themselves are well served only where an atmosphere of trust prevails. Just as the quality of the patient-physician relationship can vary as between different individuals and their personal doctors, so too may the degree of trust that prevails with respect to the medical services provided in different workplaces. It will always be incumbent upon management and labour to strive to create an atmosphere of trust in the examining physician; agreement over the identity of this person should therefore always be sought. In many workplaces, this matter can be productively worked out through labour-management deliberations. In others, for any of a number of unfortunate reasons, the matter may fester in controversy.

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[62] Letter from Mr. Arthur L. Gladstone, Senior Policy Advisor, Occupational Health and Safety Division, Ontario Ministry of Labour to the Royal Commission on Asbestos, 22 July 1983.

[63] Letter from Dr. Jerome J. Vingilis to Mr. Thomas Lederer, counsel representing the Government of Ontario with respect to the Royal Commission on Asbestos, 4 August 1982.

Such instances of controversy, whatever their source, cannot be regulated out of existence. Given this stark reality, our approach is to emphasize the importance of stressing the right of the individual employee to seek the appropriate medical examination from a physician of his own choosing. With respect to designated substance workplaces, what is necessary, in our view, is that such an examining physician should know the content of the appropriate Code for Medical Surveillance.

We conclude that the Regulation Respecting Asbestos should explicitly reflect the above considerations. This can be done by stipulating that the examining physician would be designated by the employer, save that any individual employee would have the right to be examined, at the employer's expense, by a physician of his own choosing. Such a physician, however, would have to certify that the employee's examination was conducted in accordance with the Code for Medical Surveillance. We appreciate that such explicit regulatory language could become a source of employer inconvenience in workplaces where a substantial number of workers choose to exercise their right to examination by a correspondingly large number of different physicians. But this possibility is only likely in those workplaces where an atmosphere of trust is absent. We view it as a positive incentive to management to secure the agreement of labour to the employer's choice of examining physician. We therefore recommend that:

8.11 The Regulation Respecting Asbestos should be amended to clarify that the examining physician under sections 13 to 16 shall be the physician designated by the employer, but that any employee shall have the right to be examined, at the expense of the employer, by a physician selected by the employee, provided this physician certifies to the employer that he has conducted the examination in accordance with the Code for Medical Surveillance of Asbestos Exposed Workers.

E.3 Medical Surveillance: Maintaining Records

The Regulation Respecting Asbestos requires the physician who examines a worker to maintain medical and exposure records in a secure place.[64] Where the physician is no longer able or willing to keep the records, they are to be forwarded to the Chief Physician of the Occupational Health Medical Service in the Ministry of Labour, or his designate.[65] The maintenance of these records is important for continuing studies of the health effects of designated substances, for the verification of claims and formulation of policies by the Workers' Compensation Board, perhaps to determine compliance with the control limits, and in some cases to determine whether a worker has been excessively exposed to a substance and should be treated or removed from that exposure.

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[64] Regulation Respecting Asbestos, s. 15(1).

The Regulation does not, however, indicate where geographically these records must be maintained. It appears quite possible under the Regulation to maintain these records in locations outside the province of Ontario. If this happens, then access to these records by the worker, the Ministry of Labour, or others who require them for one of the reasons stated above may be difficult. It can be virtually impossible to compel someone to produce records that are not physically located in the province.

We have become aware of instances where accessibility to records for research purposes has been problematic. In one case, when a medical researcher from the Ministry of Labour undertook an epidemiological study of the health experiences of workers at the Johns-Manville plant in Scarborough, he experienced considerable difficulty in acquiring all the necessary medical records.[66] When Bendix Automotive of Canada Limited closed its Windsor, Ontario plant, concern was expressed to us that medical records might be transferred to U.S. headquarters.[67] In fact, the Ontario Ministry of Labour has gained access to these records and is currently conducting epidemiological research with them. Nevertheless, the point remains that problems of accessibility can materialize in cases where medical records are transferred out of jurisdiction.

While companies such as Johns-Manville or Bendix have an obligation to ensure that the records of their employees are not released injudiciously,[68] workers and the scientific community have a right to some guarantee that there are records in Ontario with respect to which they can apply for access for legitimate purposes. In this regard, section 15(1) of the Regulation is deficient because it does not stipulate that the records to which it refers must be kept in Ontario.

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[65] Ibid., s. 15(2).

[66] See discussions of this issue in RCA Transcript, Evidence of Mr. Jack P. Cashman, 23 June 1982, Volume no. 44, pp. 107–114; RCA Transcript, Evidence of Dr. Paul Kotin, 29 June 1982, Volume no. 46, pp. 54–60, 118–137; and RCA Exhibit II-75, in RCA Transcript, Evidence of Dr. Paul Kotin, 29 June 1982, Volume no. 46: Correspondence between Johns-Manville and Dr. Murray M. Finkelstein, Ontario Ministry of Labour.

[67] Letter from Professor Frank C. Innes, Department of Geography, University of Windsor to the Royal Commission on Asbestos, 23 June 1980; RCA Transcript, Submission by Professor Frank C. Innes, 27 March 1981, Volume no. 6, p. 43; and International Union, United Automobile, Aerospace and Agricultural Implement Workers of America (UAW), Written submission to the Royal Commission on Asbestos, #34 [January 1981], p. 16.

[68] See a discussion of this issue in RCA Transcript, Evidence of Dr. Paul Kotin, 29 June 1982, Volume no. 46, pp. 56–60.

Beyond the fact that the Regulation fails to stipulate that records be kept in Ontario, we find more generally that the Regulation assigns too great a role to the examining physician in record maintenance. Short of the point at which, pursuant to section 15(2) of the Regulation, examining physicians who are no longer willing to keep records fulfill their obligation to forward them to the Chief Physician of the Occupational Health Medical Service of the Ministry, records will be in the keeping of physicians scattered throughout Ontario. This is a backward step from the practice whereby the chest X-rays of asbestos workers under surveillance by the Ministry's Chest Surveillance Programme were kept by the Ministry, with copies deposited in the Medical Services Division of the Workers' Compensation Board. As we point out in Chapters 13 and 14, these relatively complete records have never been systematically analyzed, but have proved useful to the Board, particularly in matters related to worker removal and rehabilitation. The record maintenance role now assigned to individual examining physicians will destroy the continuity of what has become a long and potentially valuable time series of records on workers who have been exposed to asbestos. Section 15(2) of the Regulation envisages that records can wind up being stored by the Chief Physician of the Ministry's Occupational Health Medical Service once examining physicians are no longer able or willing to keep them; we deem it highly desirable that copies of all records should be deposited with the Chief Physician at the time they are made, thereby simultaneously assuring that they are all available at one location and that this location is in Ontario.

This resolution poses an issue to which we are sensitive; the confidentiality of the medical records of the individual involved. Its complexities are so far-reaching that it was the subject of Mr. Justice Horace Krever's Commission of Inquiry into the Confidentiality of Health Information.[69] The report by that Commission specifically addressed matters of confidentiality arising from the *Occupational Health and Safety Act* and contained no fewer than seventeen recommendations on this subject. These recommendations seek to ensure stringent control of the medical records in the safekeeping of the Ministry so as to safeguard the privacy of the individual. As of the time of writing, these recommendations still awaited the response of the Government of Ontario.

We endorse the depositing of medical records with the Chief Physician of the Ministry of Labour on the explicit condition that the recommendations of the Commission of Inquiry into the Confidentiality of Health Information will be implemented. We therefore recommend that:

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[69] Ontario, *Report of the Commission of Inquiry into the Confidentiality of Health Information* (Krever Report), The Honourable Mr. Justice Horace Krever, Commissioner, 3 vols. (Toronto: Queen's Printer for Ontario, 1981), and especially chap. 33 therein.

8.12 The Regulation Respecting Asbestos should be amended so as to substitute for the role of examining physicians in maintaining the records, referred to in section 15(1), an obligation on examining physicians to forward the records or copies thereof to the Chief Physician, Occupational Health Medical Service of the Ministry of Labour. The system of control pursuant to which the Ministry maintains these records should be that prescribed by the Ontario Commission of Inquiry into the Confidentiality of Health Information.

E.4 Medical Surveillance: A Concluding Note

We have restricted ourselves, in the matter of medical surveillance for current asbestos workers, to addressing the choice of the examining physician and the maintenance of records. These are immediate and practical matters that lend themselves to formal recommendations at this time. There is a much larger question of which we are conscious, namely, the purposes that medical surveillance is intended to serve. For reasons which we give in Chapter 14, the kind of medical surveillance envisaged by the Regulation Respecting Asbestos is far more relevant to workers whose employment in the asbestos industry dates back a decade or more, when exposure levels were such as to create a significant risk of asbestosis, than it is to workers who have recently become employed. It may well be that some of the procedures prescribed by the Code for Medical Surveillance which accompanies the Regulation are excessive when applied to recently employed workers. Having raised this issue, we have concluded that the time to resolve it has not yet come. Low exposure is relatively recent, and a uniform procedure for the surveillance of all asbestos workers serves, for the time being, as an additional check on the extent to which the low exposures mandated by the Regulation Respecting Asbestos are in fact enforced.

Part IV

Asbestos in Buildings

Chapter 9 Problems of Asbestos in Buildings

A. Introduction

Part III of this Report dealt with the exposure of workers to asbestos in fixed workplaces. We now turn to the second major problem area of asbestos exposure in Ontario: the exposure of occupants and workers to asbestos fibres in buildings. In a study prepared for this Commission, Professor G. Bruce Doern reported that it was the death of a school maintenance worker, Mr. Clifton Grant, a carpenter, as a result of an asbestos-related disease, that led in part to the public pressure which resulted both in the establishment of this Commission and in the establishment of the provincial programme for controlling asbestos exposure in schools.[1]

In this chapter we will first describe the way in which asbestos was used in buildings in the past and the exposure of workers who installed that asbestos. We will then consider evidence on levels of exposure of building occupants and workers to asbestos that is already present in buildings. The particular problems involved in trying to measure asbestos hazards in buildings will be discussed. The last three sections of this chapter will evaluate the health risks faced by building occupants and the actions necessary to protect these occupants from significant risks.

This chapter will show that workers who installed or removed sprayed or pipe and boiler insulation containing asbestos without taking precautions for their own safety were exposed to high levels of asbestos fibres in the air.

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[1] G. Bruce Doern, *The Politics of Risk: The Identification of Toxic and Other Hazardous Substances in Canada*, Royal Commission on Asbestos Study Series, no. 4 (Toronto: Royal Commission on Asbestos, 1982), pp. 2.11–2.15.

Exposures of 5 to 10 fibres per cubic centimetre (f/cc) were common, and some workers were exposed to concentrations as high as 100 f/cc or more. One study has estimated that the insulation workers as a group were exposed on average over their careers to fibre levels ranging from 10 to 15 f/cc. As Chapter 5 shows, studies of these insulation workers in North America have revealed a tragic toll of disease and death resulting from this exposure.

In dramatic contrast, the exposure of building occupants to asbestos fibres during normal building use will be shown to be insignificant, whether as compared to the exposure of insulation workers in the past or as compared to the much lower exposures permitted by the recently adopted Ontario workplace control limits of 1.0, 0.5, and 0.2 f/cc for chrysotile, amosite, and crocidolite asbestos. Studies of asbestos concentrations in building air have shown that many buildings containing asbestos insulation do not exhibit fibre levels exceeding those in the outdoor air or in buildings not insulated with asbestos. Even when a building exhibits elevated asbestos fibre levels, these are still very low compared to current workplace control limits and are orders of magnitude below the level to which workers were exposed in the past. A typical building containing asbestos insulation will expose occupants to less than 0.001 f/cc of asbestos, or 1/1,000 of the current chrysotile control limit. Only a small fraction of occupant exposures in all buildings containing asbestos insulation would be as great as 0.01 f/cc of asbestos.

We will conclude that it is rarely necessary to take corrective action in buildings containing asbestos insulation in order to protect the general occupants of those buildings. On the other hand, construction, demolition, renovation, maintenance, and custodial workers in asbestos-containing buildings may be exposed to significant asbestos fibre levels and may, during their work, cause elevated fibre levels for nearby occupants. We will devote Chapter 10 to the problems of protecting these workers and of protecting occupants from possible fibre release as a result of building work. This, and not the protection of building occupants in the absence of such work, is the real challenge that asbestos insulation in buildings presents.

In this chapter we will use the word "building" in its usual sense, meaning an office, apartment, factory, warehouse, or other enclosed fixed structure frequented by people. We will see in Chapter 10 that while the vast majority of asbestos problems occur in such buildings, regulations protecting construction, demolition, renovation, maintenance, and custodial workers must extend to all construction projects including work on other structures such as subway tunnels, above-ground pipelines, and transformer vaults. Thus, in Chapter 10 we expand the definition of "building" to include any structure, enclosed or not, above ground or below, other than mines.

B. Exposure During Past Construction

A large number of products used in the construction industry contain, or have in the past contained, asbestos. These products may be divided into three categories. The first consists of asbestos-containing products used or applied in a liquid state. The release of asbestos fibres at any time from such products is unlikely, because the fibres are combined with and held down by a liquid. Examples include asbestos-containing paints and asbestos-asphalt roofing compounds. Once asbestos fibres have become impregnated with liquids such as paint or asphalt, it is very unlikely that they can become airborne again.

The second category of asbestos products is hard products, in which the asbestos fibres are firmly embedded in a solid material and are unlikely to be released during normal use. Examples include floor tiles, asbestos-cement products, hard ceiling tiles, drywall taping and joining compounds, and impregnated paper and textile products. Fibres could only be released from these products during sanding, grinding, cutting, or other work on the product during installation, renovation, or removal. Asbestos-containing floor tiles and asbestos-cement products are still used in new construction, and the exposure of workers who operate on these materials will be described in Chapter 10, Section D.2. Recommendation 10.17 prescribes safe work practices. Hard ceiling tiles and drywall taping and joining compounds might be encountered by maintenance, renovation, or demolition workers and are also encompassed by Recommendation 10.17. Asbestos-impregnated paper products were for the most part used for pipe and boiler insulation: see Section B.2 of this chapter and Chapter 10, Recommendation 10.16. Asbestos-impregnated textile products are discussed in Chapter 11, Section A. Safe work practices are dealt with in Chapter 10, Recommendation 10.17.

Finally, there are the friable, or crumbly, asbestos-containing materials such as asbestos-containing sprayed insulation and asbestos-containing pipe and boiler insulation. Friable material, defined in the United States Code of Federal Regulations, as material ". . . that can be crumbled, pulverized, or reduced to powder, when dry, by hand pressure," ranges from material which falls apart at the slightest touch to products with a relatively hard surface.[2] These materials have in the past caused substantial exposure of workers to asbestos fibres during installation and may continue to cause substantial exposure when maintenance or renovation work is performed that will disturb the material, or when the material is removed, enclosed, or encapsulated. We will first discuss sprayed insulation and then pipe and boiler insulation.

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[2] U.S., Environmental Protection Agency, *National Emission Standard for Asbestos*, 40 CFR 61.21(k).

B.1 Sprayed Asbestos-Containing Insulating Material

The spray application of asbestos fibre insulation was introduced in 1932 in Great Britain and within a few years was adopted in the United States and then in Canada. This process was originally used for thermal insulation, decorative purposes, and acoustical control, but after 1950 it was used primarily as a fireproofing to protect structural steel from heat in case of fire. The amount of asbestos-containing sprayed material installed before 1950 is a small fraction of the amount installed after 1950. Reitze et al. reported that the first use of sprayed mineral fibres for fireproofing in a large multi-storey building in the United States was in 1958.[3] Use of sprayed asbestos-containing fireproofing grew through the 1960s and far exceeded the use of asbestos-containing sprayed material for thermal, decorative, and acoustical purposes. In 1973, the spray application of asbestos material was banned in the United States by the U.S. Environmental Protection Agency (EPA). Canadian insulation manufacturers voluntarily stopped using sprayed asbestos insulation in 1973, responding in part to Regulation 419/73, section 33, under the Ontario *Construction Safety Act*, which required costly safety precautions when spraying asbestos insulation.[4] In 1978, the United Kingdom Advisory Committee on Asbestos recommended that the spraying of asbestos-containing coatings should be prohibited; and in 1981, the Health and Safety Commission issued a *Consultative Document: Asbestos Insulation and Coating — Draft Regulations*, which proposed prohibiting the spraying of any product containing more than one asbestos fibre per milligram of product. This document stated that in fact, by 1979, the process of spraying asbestos-containing insulation in the United Kingdom had been almost entirely discontinued.[5]

Sprayed asbestos-containing fireproofing was applied by either a wet or a dry process. In the wet process, chrysotile or amosite asbestos (at 5 to 30% by weight of the total formula) and some other fibres were mixed with a binder such as cement or gypsum and water and then sprayed onto the building surface. The resulting product is relatively hard and dense. The dry process was more common in Ontario than the wet process and tended to use either chrysotile, usually at 5 to 70% by weight, or amosite, usually at 50 to 90% by weight, with some other fibres and binder. In North America, crocidolite was less commonly used. It was substituted, at 50 to 90% by weight, in a small number of dry-applied applications. The dry mixture was blown through a nozzle and wetted by a water spray as it left the nozzle. This dry process resulted in a material with much lower density in place and with greater thickness than the wet process material, thus making fibre release more likely. The three types of asbestos were rarely, if ever, mixed in asbestos-containing sprayed insulation, wet- or dry-applied.[6]

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[3] William Reitze et al., "Application of Sprayed Inorganic Fiber Containing Asbestos: Occupational Health Hazards," *American Industrial Hygiene Association Journal* 33:3 (March 1972): 178–191.

[4] Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, p. 16.

[5] U.K., Advisory Committee on Asbestos, *Asbestos — Work on Thermal and Acoustic Insulation and Sprayed Coatings* (London: Her Majesty's Stationery Office, 1978), paragraph 21, p. 13; and U.K., Health and Safety Commission, *Consultative Document: Asbestos Insulation and Coating — Draft Regulations* (London: Her Majesty's Stationery Office, 1981), p. 1.

It is generally agreed that disturbing sprayed asbestos-containing insulation, whether in the process of installing it or when it is in place, generates greater airborne fibre concentrations if the asbestos is crocidolite or amosite than if it is chrysotile.[7] This is due to differences among the physical characteristics of the sprayed product and perhaps to differences among the fibre types themselves. The presence of crocidolite or amosite thus presents a greater health risk, quite apart from any difference in the toxicity of a single fibre of different types of asbestos.

In Ontario, most sprayed insulation was installed by fireproofing contractors, many of whom belonged to the Master Insulators Association. The work itself was done by insulation workers who were members of Local 95 of the International Association of Heat and Frost Insulators and Asbestos Workers. Membership in this union numbers about 700 in Ontario. Some of the high exposure experiences of these workers have been described in Local 95's written submission to the Commission.[8]

Scientific studies have confirmed that spraying asbestos-containing materials generated considerable levels of fibre exposure for the workers. Reitze et al. reported that spraying dry-process asbestos material onto a powerhouse turbine yielded fibre concentrations for the worker operating the spray nozzle ranging from 32 to 100 f/cc, while the worker charging the hopper experienced exposures of 5 to 22 f/cc.[9] The same study examined the spraying of fireproofing on a multi-storey building and found fibre counts for the worker at the nozzle of 20 to 100 f/cc and fibre counts at distances of 10 to 75 feet from the nozzle ranging from 10 to 71 f/cc during the spraying operation.[10] Thirty minutes after spraying had stopped, fibre counts ranged from 1 to 4 f/cc, and 60 minutes after the end of spraying, all counts were below 1 f/cc, probably because the dust was blown away through the open sides of the uncompleted building.

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[6] Donald J. Pinchin, *Asbestos in Buildings*, Royal Commission on Asbestos Study Series, no. 8 (Toronto: Royal Commission on Asbestos, 1982), p. 1.5; and Personal communication between Dr. Donald J. Pinchin, D.J. Pinchin Technical Consulting Ltd. and Royal Commission on Asbestos Staff, 4 February 1983.

[7] Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Evidence of Dr. John M.G. Davis, 15 January 1982, Volume no. 34, p. 28; and Personal communication between Dr. Donald J. Pinchin and Royal Commission on Asbestos Staff, 4 February 1983.

[8] International Association of Heat and Frost Insulators and Asbestos Workers, Local 95, Written submission to the Royal Commission on Asbestos, #88, 10 June 1982.

[9] Reitze et al., "Application of Sprayed Inorganic Fiber Containing Asbestos: Occupational Health Hazards," p. 181.

Skidmore and Jones reported that the nozzle worker spraying a wet process asbestos fireproofing was exposed to between 1 and 8 f/cc when care was taken to minimize dust release.[11] Barnes reported that spraying dry-process chrysotile generated fibre levels of 80 f/cc for the nozzle worker and 5 to 15 f/cc at floor levels located below the spraying operation.[12] It thus appears that insulation applications may have exposed insulation workers to fibre levels of between 5 and 100 f/cc. Other workers on the job site have experienced exposures in the range of 1 to 71 f/cc. The U.S. EPA has estimated that insulation workers encountered average exposure levels over their working lives of between 3 and 15 f/cc, with an average of 9 f/cc, while Nicholson estimated the average exposure at 10 to 15 f/cc.[13] In the Appendix to Chapter 7 of this Report, Daniels and Roberts assume an average exposure of 15 f/cc.

Where the asbestos was sprayed on an open building structure, the spray material could be carried into the outdoor environment. Selikoff, Nicholson, and Langer measured airborne asbestos concentrations in New York City at a variety of locations, including locations near construction sites where fireproofing was being sprayed. The asbestos concentrations ranged from an average of less than 10 nanograms per cubic metre (ng/m^3) of air[14] in Queens and Staten Island to an average of 30 ng/m^3 of air in Manhattan.[15] Within one-quarter mile of a spray fireproofing site, the average asbestos concentration was 60 ng/m^3, with a range up to 375 ng/m^3. Up to one-half mile away, the average concentration was 25 ng/m^3, with a range up to 54 ng/m^3. The report concluded that ". . . at least in the areas sampled, there is a background of chrysotile contamination of the ambient air and that this may be higher about construction sites in urban areas."[16]

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[10] Ibid., p. 182.

[11] J.W. Skidmore and J.S. Jones, "Monitoring an Asbestos Spray Process," *Annals of Occupational Hygiene* 18:2 (September 1975): 154.

[12] R. Barnes, "Asbestos Spraying, An Occupational and Environmental Hazard," *Medical Journal of Australia* 2:16 (16 October 1976): 600.

[13] U.S., Environmental Protection Agency, Office of Toxic Substances, *Support Document for Final Rule on Friable Asbestos-Containing Materials in School Buildings: Health Effects and Magnitude of Exposure* (Washington, D.C.: U.S. Environmental Protection Agency, January 1982), p. 90. See also, William J. Nicholson, *Dose-Response Relationships for Asbestos and Inorganic Fibers* (New York: Environmental Sciences Laboratory, Mount Sinai School of Medicine of the City University of New York, 15 February 1981), p. 26.

[14] The mass, or weight, of asbestos in the air may be expressed in terms of nanograms, or billionths of a gram, of asbestos per cubic metre of air. Mass measurements are not easily converted to fibre count equivalents. See Section D.3 of this chapter.

[15] Irving J. Selikoff, William J. Nicholson, and Arthur M. Langer, "Asbestos Air Pollution," *Archives of Environmental Health* 25:1 (July 1972): 10–11.

The spraying of asbestos-containing insulation materials has been discontinued in Ontario under strict regulations for the spray process. We will show in Chapter 10 that the presence of asbestos-containing insulation in a building requires constant vigilance to protect building workers from excessive asbestos fibre levels. There we will recommend that asbestos insulation should be removed from any building before that building is demolished, imposing an additional cost on the owner. Sprayed insulation in buildings not only poses a risk for workers on the construction site, but poses possible risks for demolition, renovation, maintenance, and custodial workers in the building for its entire life. As well, we have seen that the process of installing sprayed asbestos-containing insulation tends to pollute the ambient air. Notwithstanding the regulations now in place, we believe that the application of sprayed insulation materials containing asbestos, wet or dry, should be prohibited.

This is contemplated in Ontario's Proposed Regulation Respecting Asbestos on Construction Projects (PRRACP), in section 9(2): "No material containing asbestos shall be applied with a pressure spray system."[17] The prohibition, however, should be framed in such a manner that it can be effectively enforced. As it stands, the wording of the proposed regulation could undermine its enforcement because it is impossible to analyze effectively to a zero concentration, and because many insulation materials contain trace amounts of asbestos as a material contaminant. In light of this, the U.S. EPA has prohibited the spraying of insulation containing more than 1% asbestos by weight.[18] We believe that 1% is the practical lower limit on the quantitative analysis of bulk material. Accordingly, we recommend that:

9.1 The Ministry of Labour should prohibit the spraying of friable material containing more than 1% asbestos on a dry-weight basis.

Non-asbestos materials were used for fireproofing and insulation before 1950, and a variety of new non-asbestos fireproofing and insulation materials, including sprayed materials, are available at reasonable cost.

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[16] Ibid., p. 11.

[17] Ontario, Ministry of Labour, Occupational Health and Safety Division, "Proposed Regulation Respecting Asbestos on Construction Projects and Related Codes," presented at a Public Meeting, 17 January 1983, Toronto, s. 9(2). (Mimeographed.) This "Proposed Regulation. . ." is a revision of the "Notice of Proposed Regulation: Designated Substance — Asbestos on Construction Projects," *The Ontario Gazette*, vol. 115-33, Saturday, 14 August 1982, pp. 3194-3197.

[18] U.S., Environmental Protection Agency, *National Emission Standard for Asbestos*, 40 CFR 61.22(e).

Major substitutes for asbestos fibres include mineral wool and cellulose fibre. Cellulose fibre is frequently made from processed paper. These fibres are not highly durable in the lung and health hazards have not been found to result from the manufacture or use of this product.[19] In Chapter 6 we review the evidence on the health effects of breathing dust from mineral wool. Some evidence has suggested that mineral wool is no more hazardous than fibreglass.[20] Other studies have found that respiratory mortality is increased by exposure to mineral wool, but that the resulting standard mortality ratios (SMRs) are far lower than for asbestos.[21] We conclude, in Chapter 6, that the low historical disease record from exposure to mineral wool might reflect low historical worker exposures, and that processes that exposed workers to higher than previously experienced concentrations of smaller diameter fibres might result in serious disease. The absence of a completely clean bill of health for these materials suggests the wisdom of imposing standards that would avoid excessive worker exposure. We accordingly recommend that:

9.2 The Ministry of Labour should assess, sponsoring studies where appropriate, the health risks associated with the fibres used as substitutes for asbestos in sprayed insulation, with a view to determining what regulations, if any, might be appropriate for the use of these substitutes.

B.2 Pipe and Boiler Insulation

Historically, a variety of products were used for insulating pipes and boilers. These included preformed thermal insulating sections or slabs, asbestos-cement compounds, corrugated asbestos paper, rope lagging, and asbestos tape.

Preformed thermal insulating sections or slabs were widely used for insulating or "lagging" pipes, boilers, and furnaces from the mid-1920s through 1972.[22] While the sprayed insulation discussed above needs no more structural integrity than that necessary to attach it to a surface, preformed thermal insulating sections maintain their shape during shipping and installation. At the same time, while preformed thermal insulating sections are structurally solid, they do not have the strength and durability of asbestos-cement products. A variety of fibre types and binding materials were used in manufacturing these sections, with asbestos usually representing 15% by weight of the product, and in any event not exceeding 80% by weight of the product. Magnesium carbonate and calcium silicate were the two most common binders.

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[19] GCA Corporation, "Asbestos Substitute Performance Analysis," draft revised final report prepared by Nancy Krusell and David Cogley for U.S. Environmental Protection Agency, GCA-TR-81-32-G (Bedford, Mass.: GCA Corporation, February 1982), p. 305.

[20] Douglas P. Fowler, "Occupational Exposures to Mineral Wool," in *Proceedings of the National Workshop on Substitutes for Asbestos*, Arlington, Virginia: 14–16 July 1980, EPA-560/3-80-001 (Washington, D.C.: U.S. Environmental Protection Agency, November 1980), p. 400.

[21] Philip E. Enterline, Gary M. Marsh, and Nurtan A. Esmen, "Respiratory Disease Among Workers Exposed to Man-Made Mineral Fibers," *American Review of Respiratory Disease* 128:1 (July 1983): 1–7.

[22] Personal communication between Mr. Ian Dewar, Dewar Insulations Inc. and Royal Commission on Asbestos Staff, 8 March 1983.

Corrugated asbestos paper was sometimes made into preformed insulating sections for lower-temperature pipe insulation. This paper consists of almost pure asbestos.

While preformed sections were used on pipes and boilers, irregular fittings, such as elbows, Ts, and valves could not be covered by standardized preformed shapes. These fittings were, and to a small extent still are, covered by asbestos-cement compounds, rope lagging, or asbestos tape. These asbestos products were often used even on building projects where the insulating sections did not contain asbestos. Thus, while preformed insulating sections account for the greater quantity of asbestos in pipe and boiler lagging, the asbestos products used in irregular fittings may occur much more frequently and thus appear in buildings that use non-asbestos insulation for all other purposes.

Asbestos-cement compounds, sometimes referred to as asbestos-insulating cement, are powders with a high content of a commercial grade of asbestos fibres called "shorts" and with, in some cases, Portland cement used as a binder. They are mixed with water on the job site and applied in a dense mass by hand. Oddly, asbestos-insulating cement often contains no cement. The addition of water to the asbestos creates a material that retains its structural integrity when dried. Asbestos-insulating cement is quite different from what are known as asbestos-cement products, which always contain cement, are manufactured in a factory, are very hard, and are not used for insulating pipes and boilers. Frequently, asbestos-cement compounds, once installed, are less friable than insulating blocks or corrugated paper, because they are more dense. The three joint products — asbestos-cement compounds, rope lagging, and asbestos tape — are generally almost pure asbestos. However, once in place these products do not generate high dust levels.[23]

In his study for this Commission, Dr. Donald J. Pinchin noted that a higher proportion of Ontario buildings will contain asbestos-containing pipe coverings than sprayed asbestos-containing fireproofing.[24] The major suppliers in Ontario of pipe and boiler insulation were Atlas Asbestos Company, Johns-Manville Canada, Holmes Insulations Ltd., and Philip Carey Company. Saheed et al. have reported that Atlas and Holmes used mainly amosite while Johns-Manville used crocidolite until 1960, followed by chrysotile from 1960 to 1973. By 1973, all four companies had stopped using asbestos in their pipe and boiler insulation products.[25]

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[23] Ibid.

[24] Pinchin, *Asbestos in Buildings*, p. 3.18.

Nicholson, reporting on Reitze's study of dust concentrations occurring in pipe and boiler insulation work, found that with the standard work practices of the 1960s, workers engaged in cutting and applying new insulating block or sections of pipe covering were exposed to 5.2 f/cc with good ventilation, and as much as 11.5 f/cc with poor ventilation. Workers engaged in mixing and applying asbestos-insulating cement and cloth covering to insulation material experienced 2.5 and 4.6 f/cc with good and poor ventilation respectively. Improved ventilation reduced these exposure levels by a factor of 2 or more.[26]

Once the insulation is installed, the heat from the pipe or boiler causes the insulation to become increasingly fragile, and thus more friable (or crumbly) over time. Accordingly, removal of old insulation could cause more dust than arose in the original installation. From the above we conclude that asbestos-containing pipe and boiler insulation presents a continuing hazard to building workers.

As with sprayed insulation, much of the asbestos-containing pipe and boiler insulation work in Ontario was performed by firms belonging to the Master Insulators Association, using workers from Local 95 of the International Association of Heat and Frost Insulators and Asbestos Workers.

The Ministry of Labour's Proposed Regulation Respecting Asbestos on Construction Projects does not appear to regulate the use of asbestos in pipe and boiler insulation. A draft regulation, "Asbestos Insulation and Coating," was prepared in the United Kingdom in 1981. This proposed regulation would prohibit the use of asbestos in pipe and boiler insulation.[27] Because of the continuing problem of worker exposure in buildings containing friable asbestos-containing pipe and boiler insulation, we recommend that:

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[25] W. Saheed et al., "Distribution and Concentrations of Asbestos Dust in Commonplace Construction Activities," a project conducted jointly by the University of Toronto, Department of Preventive Medicine and Biostatistics and the Construction Safety Association of Ontario, Research and Development Department, Toronto, 1978 (Mimeographed.); and Personal communication between Mr. Ian Dewar and Royal Commission on Asbestos Staff, 8 March 1983.

[26] William J. Nicholson, "Proposed Standard for Workers is Questioned," *Insulation Hygiene Progress Reports* 3:2 (Summer 1971): 3.

[27] U.K., Health and Safety Commission, *Consultative Document: Asbestos Insulation and Coating — Draft Regulations*, p. 3.

9.3 The Ministry of Labour should ban the application of friable materials containing more than 1% asbestos by weight as insulation for pipes and boilers.

It appears that a number of asbestos-free materials are available for pipe and boiler insulation, including fibreglass and mineral wool products. Some of these materials were used before asbestos became popular, and others have been developed more recently. In particular, preformed fibreglass and calcium silicate insulation with non-asbestos fibres appear to enjoy widespread popularity for pipe and boiler insulation today. The substitutes seem to perform adequately without substantial cost increases over asbestos products. We review in Chapter 6 the health evidence on mineral wool and fibreglass products. It is not known whether other substitute materials for asbestos-containing pipe and boiler insulation pose health concerns, and we accordingly recommend that:

9.4 The Ministry of Labour should identify the compounds used as substitutes for pipe and boiler insulation, with a view to assessing any health risks associated with these substitutes and to determining what regulations, if any, might be appropriate for the use of these substitutes.

C. Measurement of Asbestos Fibre Levels in Buildings and Outdoors

In examining exposure levels of occupants and workers in buildings, it is helpful to know something about how these levels are measured. To determine the asbestos fibre concentration in the air, an air sample is drawn through a filter. The material on the filter is then analyzed using either an optical microscope or an electron microscope, and the concentration of material in the air where the sample was taken is estimated.

Three kinds of measurements may be used to quantify the asbestos concentration in the air in buildings. The first is the measurement by the phase contrast microscope (PCM), a form of optical microscope, of the number of fibres (both asbestos and non-asbestos) longer than 5 microns[28] per cubic centimetre (f/cc) of air. This is discussed in Chapter 7 where it is referred to as the "membrane filter method" and is the method used by the Ministry of Labour to specify and enforce the current control limits in a fixed workplace. The second measurement method is the use of the transmission electron microscope (TEM) to determine the number of asbestos fibres per cubic centimetre of air. The TEM may be used to count asbestos fibres of all lengths, or only those fibres longer than 5 microns. The third method uses a TEM fibre count to estimate the weight or mass of asbestos fibres in the atmosphere. The mass is expressed in nanograms (one-billionth of a gram) per cubic metre of air (ng/m^3). We will refer to measurements using all three of these methods.

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[28] While the correct technical term is now micrometre, we will use the more familiar term micron.

In Chapter 7 we describe the membrane filter method using PCM to analyze airborne asbestos fibre levels in fixed workplaces. The PCM does not provide a method for identifying what type of fibre has been observed, so that it cannot distinguish between asbestos fibres and other fibres. The PCM yields a fibre count only, *not* a count of asbestos fibres. Fibre identification cannot be achieved without the use of another form of optical microscopy, polarized light microscopy (PLM), or transmission electron microscopy (TEM).

Inability to identify fibre types is not usually a problem in workplaces such as asbestos manufacturing plants, or in buildings during the removal of friable asbestos insulation, because in these instances one can reasonably assume that most fibres in the atmosphere are in fact asbestos fibres. However, in buildings or in the ambient environment where asbestos may represent anywhere from 0 to 100% of the fibres in the air, the PCM is a relatively unreliable means of determining asbestos fibre concentrations. Furthermore, in buildings or outdoors, the majority of the fibres are too thin to be seen by the PCM. This problem arises in the context of fixed workplaces too, but the proportion of thin fibres is generally smaller in fixed workplaces.

Thus, the result of an optical fibre count performed on a building or an environmental sample can be challenged in two ways. If the fibre count is low, it does not establish that asbestos is not present, since there may have been asbestos fibres with diameters too small for detection. If the fibre count is high, it may be argued that few if any of the fibres were actually asbestos, and that most belonged to some other fibrous species. Only when the fibre count is high enough for reliable counting by PCM, perhaps 0.5 f/cc, and when there is reason to believe that most fibres in the air are asbestos, as during asbestos insulation removal, is PCM air monitoring useful in buildings. Even then it leaves thin fibres uncounted.

Chatfield has reported that it is widely accepted that the PCM method is not well suited to the measurement of asbestos fibres in the ambient atmosphere: "The U.S. National Institute for Occupational Safety and Health recommends against the use of optical microscopy techniques for monitoring of ambient atmosphere. Many others also reject the use of PCM for environmental measurements, whether inside or outside buildings, and the U.S. Environmental Protection Agency has specified a method based on transmission electron microscopy (TEM) for asbestos measurements in ambient atmospheres."[29]

The transmission electron microscope is the preferred instrument for analyzing the low asbestos fibre content of building or outdoor air samples. A modern TEM has a resolution of about 0.2 nanometres, which is more than adequate for resolving the smallest fibres of chrysotile, which are about 40 nanometres in diameter.[30] The TEM image also permits observation of some of the internal structure of each fibre. Several methods of identifying the type of fibre observed are available on the TEM, as will be indicated later.

Although the TEM is a sophisticated and precise instrument, capable of observing and identifying very small asbestos fibres, its use for determining airborne asbestos concentrations is beset with difficulties. These difficulties lend uncertainty to the resulting fibre counts. The problems inherent in using a TEM may be seen from a brief review of the procedure for preparing and analyzing TEM samples.

Two types of filters may be used for gathering air samples for TEM analysis, each appropriate for a particular preparation method. The Nuclepore filter has a flat surface and is used for "direct preparation," in which the exposed filter is given a carbon-coating on top which captures the fibres for examination in the TEM. Chatfield, in his work at the Ontario Research Foundation, uses this method for building and environmental samples. This method has the advantage that fibres are observed in the position in which they were first captured on the filter with a minimum of disturbance. A disadvantage is that if the filter is handled roughly in the field, before carbon-coating, fibres may be dislodged and lost.

Alternatively, one may use a cellulose ester (Millipore) filter which has a spongy surface and is used for "indirect" preparation. While various indirect preparation methods have been used, many have now been discredited because they lost asbestos fibres or because asbestos fibre contamination was introduced during the process. Some researchers use an ashing method in which the cellulose filter is ashed, which removes all particulates except indestructible material which is predominantly asbestos. The ash is then dispersed in water and refiltered for direct preparation. This ashing and redispersal provides a uniform fibre distribution on the surface of a filter that is well suited for TEM work. However, fibre bundles may be broken up into more numerous fibres so that the resulting fibre count may be higher than it would have been with direct preparation. There is also some concern that fibres may be lost or added if the process is not performed very carefully.

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[29] Eric J. Chatfield, *Measurement of Asbestos Fibre Concentrations in Ambient Atmospheres*, Royal Commission on Asbestos Study Series, no. 10 (Toronto: Royal Commission on Asbestos, 1983), p. 18.

[30] Eric J. Chatfield, "The Problems of Measurement of Asbestos," in Ontario, Royal Commission on Asbestos, *Proceedings of The Royal Commission on Asbestos Second Public Meeting, Friday, December 12, 1980*, reported by Lydia Dotto (Toronto: Royal Commission on Asbestos, 1981), Appendix A, p. 12. The structures of amosite and crocidolite fibres are different from that of chrysotile, so there is no minimum irreducible fibre size, as there is for chrysotile.

Once a sample is prepared, using either of the above methods, the fibres may be counted on a TEM in a manner similar to that used for optical fibre counting, except that each fibre is subjected to an identification process so that only asbestos fibres are counted. Because building and outdoor samples may include a large proportion of non-asbestos fibres, the identification step is very important.

Chrysotile may be identified by appearance (morphology) alone if there is no suspicion that materials of similar appearance are present. Identification of individual fibres can also be achieved by using selected area electron diffraction (SAED), in which a pattern characteristic of the crystal structure of the fibre is produced in the microscope. However, since the crystal structures of the amphiboles — tremolite, actinolite, crocidolite, anthophyllite, and amosite — are practically identical, it is not possible to distinguish among these minerals using SAED alone. Discrimination among the amphiboles can only be achieved reliably by measurement of the chemical composition of the fibres. Energy dispersive x-ray analysis (EDXA) equipment, which can be incorporated into the TEM, allows these composition data to be obtained for each fibre in addition to obtaining the diffraction patterns. Because outdoor and building air samples may contain a wide variety of fibre types it is important to examine carefully the fibre identification method used.

The results of a TEM analysis of an air sample can be presented as a fibre concentration measured in fibres per cubic centimetre. Because the length and diameter of each fibre is recorded, the fibre count can be presented as the total number of fibres per cubic centimetre observed and as the number of fibres longer than 5 microns per cubic centimetre observed. Any time that fibres longer than 5 microns are found, many short fibres will also be present, so the total fibre count is often 10 or 100 times as great as the count of fibres over 5 microns. As well, because the TEM can detect fine fibres it may produce a count of fibres longer than 5 microns that is much greater than the corresponding count obtained by PCM. (See Section D.3 of this chapter regarding the conversion of TEM fibre counts to equivalent optical counts.)

The same data could be used to determine the mass of asbestos observed. The length and diameter of a fibre can be used to determine fibre volume. Once the fibre is identified, its density is known, and multiplying the fibre volume by density yields the mass of the fibre. While a fibre count is usually presented in fibres per cubic centimetre, a mass measurement is presented in nanograms per cubic metre of air. A nanogram is one-billionth (10^{-9}) of a gram. Note that even when mass measurements of asbestos concentrations in the air are presented, they are derived not from weighing the asbestos, but rather from estimating its weight based upon the dimensions and density of every fibre counted.

Chatfield has reported that there is as yet no international agreement on the appropriate method for measuring asbestos fibre concentrations in the ambient or building air.[31] While work is currently in progress to develop standardized methods, they may not be agreed upon for several years. In the meantime, we must rely upon data from different researchers, prepared by very different methods, which are not easily comparable to each other. Thus, although the TEM may be the best equipment for analyzing ambient or building air samples, the inter-laboratory differences in analytical procedure cause considerable inter-laboratory differences in reported airborne asbestos concentrations.

D. Exposure of Occupants and Workers in Existing Buildings

The principal source of asbestos fibres in the air of existing buildings is the release of fibres from damaged or disturbed friable insulating material. Sawyer and Spooner have identified three modes of contamination of building air by asbestos fibres from such material. They are:

(i) fallout or erosion, in which asbestos fibres fall from the insulating material or are blown loose from it by air flow;

(ii) physical contact with the surface of the material, which breaks some material loose; and

(iii) secondary dispersion or re-entrainment, which is the disturbance of asbestos fibres which have already fallen onto building surfaces, by normal building activity or building cleaning.[32]

It appears that fallout and erosion generally produce minimal levels of fibre concentration in building air. Impact can produce higher fibre levels, though generally for very short periods of time. Secondary dispersion can cause significant fibre levels, particularly if substantial amounts of asbestos material have fallen onto the floor or other building surfaces.

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[31] Chatfield, *Measurement of Asbestos Fibre Concentrations in Ambient Atmospheres*, p. 95.

[32] Robert N. Sawyer and Charles M. Spooner, *Sprayed Asbestos-Containing Materials in Buildings: A Guidance Document, Part 2*, prepared for the U.S. Environmental Protection Agency, March 1978, EPA-450/2-78-014 (Washington, D.C.: U.S. Environmental Protection Agency, March 1979), pp. 1-2-5, 1-2-7.

We will now review the available data on the fibre levels that building occupants and building workers may experience.

D.1 Studies Using the Optical Microscope

Pinchin summarized the results of several optical microscopy studies of building atmospheres.[33] He reported that the South Australian Health Commission found levels of airborne fibre in buildings containing sprayed asbestos-containing material under normal conditions to be very similar to fibre levels in buildings without asbestos; the average background fibre level in buildings without sprayed asbestos was reported as 0.03 f/cc, with a maximum amount of 0.05 f/cc. Only 2 asbestos-containing buildings out of 11 in this study yielded fibre levels of 0.1 f/cc or greater. However, when maintenance employees in these buildings moved ceiling tiles or worked above the ceiling in a ceiling space containing sprayed asbestos, they were exposed to fibre levels which rose to approximately 1 f/cc, with a maximum of 5.45 f/cc, using personal samples on the maintenance workers.[34] It should be emphasized that, because of the limitations of the optical microscope, there is no way of knowing what proportion of these observed fibres were asbestos and what proportion were other dusts.

Sawyer found results similar to those of the South Australian Health Commission, with fibre levels of 0.2 f/cc and below in a library during normal activity or no activity. During cleaning of books, replacing of light bulbs, sweeping, dusting, and work on hanging lights, dust levels ranged from 1 f/cc to a maximum of 17.7 f/cc. The library building had been sprayed with a mixture of asbestos (15% by weight of the mixture being chrysotile), fibreglass, and cement binder, much of which was within reach of library users and in poor condition.[35] Again, it is not known what portion of the recorded fibres were in fact asbestos fibres.

Other optical microscope studies surveyed by Pinchin showed fibre levels below 0.3 f/cc with normal activity and above 2 f/cc during high activity, disturbance of the asbestos-containing insulation, and cleaning operations. Considering all the data, Pinchin concluded that under normal conditions fibre levels are quite low in buildings unless the sprayed material is in unusually poor or exposed condition, and even then low fibre levels are sometimes measured. However, maintenance activities and dry cleaning methods consistently raise airborne fibre levels substantially, although optical microscope methods do not allow the determination of the asbestos content of these higher dust levels.

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[33] Pinchin, *Asbestos in Buildings*, Table III, pp. 1.11-1.12.

[34] D.J. Hamilton, "Asbestos Insulation in South Australian Buildings: Hazard Assessment and Control Experience," draft paper prepared for the South Australian Health Commission [Canberra], 1980. (Mimeographed.) Note that the detection limit for optical measurement is about 0.1 f/cc, so the reported concentrations below this level should be regarded as subject to considerable uncertainty.

[35] Robert N. Sawyer, "Asbestos Exposure in a Yale Building: Analysis and Resolution," *Environmental Research* 13:1 (February 1977): 153.

It must be emphasized that optical microscopy fibre level measurements do not distinguish between asbestos and non-asbestos dust. Accordingly, optical microscopy results indicate the highest possible levels of asbestos fibres large enough to be seen by the optical microscope, rather than actual asbestos fibre levels. There are, however, significant limits on the kinds of fibres that are visible under an optical microscope; in addition to not counting fibres shorter than 5 microns, the optical microscope misses fibres thinner than about 0.3 microns.

D.2 Studies Using the Transmission Electron Microscope

There are several TEM studies of airborne asbestos fibre levels in buildings. Sebastien et al., using TEM methods, compared asbestos fibre levels in the outdoor air in Paris, in buildings without asbestos, and in buildings with sprayed asbestos insulation. Sebastien defined "abnormally high" asbestos concentrations as those above the concentration found in 99% of his outdoor samples. Applying this definition to his 161 outdoor samples, he concluded that fibre levels above 7 ng/m^3 were "abnormally high." This is consistent with his analysis of 7 buildings without sprayed asbestos insulation in which he found an average of 1.9 ng/m^3. The highest single reading in a building that did not contain asbestos was 12 ng/m^3.[36]

Sebastien analyzed 21 buildings that contained sprayed asbestos insulation, collecting 127 air samples. In 9 out of the 21 buildings, the maximum concentration of asbestos fibres was less than 7 ng/m^3 and therefore not "abnormally high." Of the remaining 12 buildings, there was at least one sample above the 7 ng/m^3 level in each building. The 8 highest samples, found in 4 buildings, exceeded 100 ng/m^3, and 6 samples exceeded 400 ng/m^3. The average (arithmetic mean) asbestos fibre concentration in the 21 buildings ranged from 1 to 70 ng/m^3, with the worst single measurement at 751 ng/m^3, and 50% of all samples below 5 ng/m^3. These data are summarized in Table 9.4 in Section D.4 of this chapter. An analysis of air in various buildings during normal use demonstrated that "abnormally high" asbestos fibre levels were more common in buildings with soft, fibrous asbestos than with cementitious asbestos material, and that fibre levels were particularly elevated in buildings with obvious material deterioration or with exposed material and high levels of activity.

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[36] Patrick Sebastien et al., *Measurement of Asbestos Air Pollution Inside Buildings Sprayed with Asbestos*, translation of document prepared for the Government of France, Ministry of Health and Ministry for the Quality of Life Environment, 1977, EPA-560/13/80-026 (Washington, D.C.: U.S. Environmental Protection Agency, August 1980), p. 59.

Nicholson, Rohl, and Weisman used rather different analytical techniques to examine outdoor air, buildings without asbestos, and buildings with sprayed asbestos insulation.[37] (See Table 9.4.) Of 23 samples of outdoor urban air, Nicholson found 19, or 83%, below 20 ng/m^3 of asbestos. Twenty-two readings, or 96%, were below 50 ng/m^3, with a maximum reading of 87 ng/m^3. It is interesting to note that in comparison with Sebastien, 99% of Nicholson's outdoor readings would not be below 7 ng/m^3, but below 87 ng/m^3. This means that Sebastien's definition of "abnormally high" asbestos concentrations, when applied to Nicholson's data, would involve readings of above 87 ng/m^3. Nicholson took 12 samples in 2 buildings that did not contain sprayed asbestos insulation. Here he found 11 samples, or 92%, below 20 ng/m^3, and a maximum concentration of 42 ng/m^3. Thus, the asbestos fibre concentration measured by Nicholson in the air of buildings without asbestos-containing insulation was above zero, but still somewhat lower than that measured in the outdoor air.

In the same study, Nicholson examined 17 buildings containing sprayed wet-applied or dry-applied asbestos. Of 28 samples taken in buildings with wet-applied asbestos, 26, or 93%, contained less than 20 ng/m^3 of asbestos. Most of these readings were in the 5 to 20 ng/m^3 range. Only one sample contained more than 50 ng/m^3 and it measured 180 ng/m^3. Nicholson concluded that buildings with sprayed wet-applied asbestos do not have fibre levels significantly above outdoor concentrations. However, in buildings containing sprayed dry-applied asbestos, of 53 samples, 28, or 53%, contained less than 20 ng/m^3 of asbestos, again mostly in the 5 to 20 ng/m^3 range. Forty-four samples, or 83% of the total, were less than 50 ng/m^3; while 2 samples, or 4%, exceeded 200 ng/m^3. The highest measurement was 830 ng/m^3 in a generator room, not frequented by general building occupants. The next highest measurement was 210 ng/m^3. Nicholson concluded that the airborne asbestos fibre levels in buildings with dry-applied asbestos spray are significantly above those in buildings without asbestos or in the outdoor air.

Sawyer and Spooner reported on a small number of measurements of asbestos fibre levels in buildings containing exposed sprayed asbestos-containing insulation, using TEM methods.[38] Three air samples in a Connecticut office building during normal activity yielded an average concentration of 79 ng/m^3 and a maximum of 110 ng/m^3. Studies in New York office buildings containing asbestos-containing insulation yielded an average in the building with the lowest asbestos levels of 2.5 ng/m^3 and in the most contaminated building of 200 ng/m^3. No details are available on the number of samples, the number of buildings, or the method of measurement. Sawyer and Spooner also reported on fibre levels measured in buildings with custodial activity that disturbs settled asbestos fibres, revealing considerably higher concentrations of 643 ng/m^3 in one building, and 296 ng/m^3 in another.[39] In general, the readings presented by Sawyer were higher than those of either Sebastien or Nicholson, although less information about the method of taking the samples and analyzing them was provided by Sawyer than by the other researchers.[40]

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[37] William J. Nicholson, Arthur N. Rohl, and Irving Weisman, *Asbestos Contamination of the Air in Public Buildings*, EPA-450/3-76-004 (Research Triangle Park, N.C.: U.S. Environmental Protection Agency, October 1975), Table 4, p. 24.

[38] Sawyer and Spooner, *Sprayed Asbestos-Containing Materials in Buildings: A Guidance Document*, p. I-2-9.

Pinchin conducted a study for this Commission of airborne asbestos fibre levels in Ontario buildings, in which he collected air samples that were analyzed by Chatfield at the Ontario Research Foundation, using a TEM. Table 9.1 reports on asbestos fibres detected in 19 buildings, counting both asbestos fibres of all lengths and asbestos fibres longer than 5 microns. While all buildings contained asbestos insulation, only 5 yielded detectable asbestos fibres longer than 5 microns. All fibre levels reported were extremely low, whether measured in ng/m^3 or in f/cc, despite the high resolution of the electron microscope. The highest concentration of fibres longer than 5 microns is 0.003 f/cc. The fibre concentrations reported in Table 9.1 may be compared to those in the outdoor environment reported in Chapter 11; the building concentrations are similar to prevailing outdoor fibre concentrations.

Since the TEM can detect fibres not seen with an optical microscope, these fibre levels can safely be interpreted as less than 1% of both Ontario's recently adopted 1 f/cc workplace control limit for chrysotile and the 0.5 f/cc control limit for amosite. On the other hand, in all of these buildings there was no particular disturbance to the asbestos-containing material, so that peak concentrations could be generated considerably above the levels shown in Table 9.1.

Table 9.2 shows the results of monitoring the air in Ontario buildings containing asbestos insulation while work was being performed, using great care to minimize fibre release. Both the optical microscope and the TEM were used for analysis. The top half of the table indicates fibre levels during inspection above a suspended ceiling, and the bottom half indicates fibre levels during minor maintenance or renovation of a suspended ceiling. The optical microscope counts are all quite low when compared with the recently adopted 1 f/cc control limit for chrysotile, despite the fact that fibres other than asbestos may well be included in these counts. These

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[39] Ibid.

[40] For a summary of these three studies, see Pinchin, *Asbestos in Buildings*, Table IV, p. 1.17.

Table 9.1

Results of Air Monitoring Using Transmission Electron Microscopy in Nineteen Ontario Buildings

Building No.	Number of Samples	Asbestos Fibres of All Lengths f/cc	Asbestos Fibres of All Lengths ng/m³	Asbestos Fibres >5.0 μm f/cc	Asbestos Fibres >5.0 μm ng/m³	Asbestos Type
1	4	0.0007	0.59	0.001	0.28	amosite
2	5	0.006	3.0	0.0*	0.0	chrysotile
3	1	0.004	0.026	0.0	0.0	chrysotile
4	3	0.003	0.067	0.0	0.0	amosite
5	5	0.0	0.0	0.0	0.0	chrysotile
6	3	0.015	1.7	0.001	0.067	amosite
7	5	0.01	0.017	0.0	0.0	chrysotile
8	3	0.004	0.31	0.0	0.0	chrysotile
9	2	0.20	0.004	0.0	0.0	chrysotile
10	4	0.01	0.045	0.0	0.0	chrysotile
11	7	0.009	11.	0.001	10.6	chrysotile
12	3	0.023	3.3	0.003	1.2	amosite
13	2	0.010	0.027	0.0	0.0	chrysotile
14	4	0.016	2.7	0.002	1.5	amosite
15	1	0.059	8.0	0.0	0.0	chrysotile
16	2	0.003	0.012	0.0	0.0	chrysotile
17	4	0.027	0.70	0.0	0.0	chrysotile
18	1	0.010	0.13	0.0	0.0	amosite
19	4	0.001	0.016	0.0	0.0	amosite

Note: *The figure 0.0 indicates that no fibres were detected; the concentration is therefore below the detection limit for that sample.

SOURCES: Data in last column of this table are from: Personal communication between Dr. Donald J. Pinchin, D.J. Pinchin Technical Consulting Ltd. and Royal Commission on Asbestos Staff, 14 December 1982.

Other data in this table are adapted from: Donald J. Pinchin, *Asbestos in Buildings*, Royal Commission on Asbestos Study Series, no. 8 (Toronto: Royal Commission on Asbestos, 1982), Table II, p. 6.7.

Table 9.2

Air Monitoring Results During Inspection or Maintenance

Sample Location	Personal or Area	Optical Microscopy (PCM) f/cc	Transmission Electron Microscopy			
			Total Asbestos Fibres		Asbestos Fibres >5.0 μm	
			f/cc	ng/m³	f/cc	ng/m³
Inspection above suspended ceiling						
Inspector	P	0.31	0.152	14.7	<0.02	—
Observer	P	0.15	0.39	460.7	0.016	420
Background	A	<0.009	0.086	3.3	<0.008	—
Background	A	0.009	0.092	1.6	<0.008	—
Maintenance, renovation above suspended ceiling						
Worker above ceiling	P	0.17*	370	6,400	12.0	1,100
Assistant	P	0.06	2.68***	104***	<0.07***	—
Close to work site	A	0.009*, **	0.179	2.6	<0.02	—
Far from work site	A	<0.005**	0.118	8.0	<0.008	—

Notes: *Average of 2 samples.
**These figures are probably below the limit of reliable detection.
***Approximate.

SOURCE: Adapted from Donald J. Pinchin, *Asbestos in Buildings*, Royal Commission on Asbestos Study Series, no. 8 (Toronto: Royal Commission on Asbestos, 1982), Tables I and II, pp. 7.5, 7.8.

optical counts are also low in comparison with those found by other researchers and discussed earlier in this section, probably because of the great care to minimize fibre release with which the work was performed.

Turning to the TEM results, and considering only asbestos fibres longer than 5 microns, the asbestos concentration is negligible in 6 out of 8 cases. Only one of the readings, that of the personal sample on the maintenance worker above the ceiling, yields a concentration of fibres, longer than 5 microns, that exceeds the 1 f/cc workplace control limit for chrysotile. As we note above, the electron microscope can detect many fibres not observed under the optical microscope, and the corresponding optical count is below the 1 f/cc standard for chrysotile. If the mass measurements of asbestos fibres of all lengths are considered, 5 of the 8 readings are above the 7 ng/m^3 level established as "abnormally high" for outdoor air by Sebastien, although this comparison may not be meaningful, as Sebastien used different analytic methods. With the exception of the personal sample on the worker above the ceiling, these TEM results are generally low compared to results of other studies, perhaps because of the care with which the maintenance work was performed, or perhaps because care was taken to count only identified asbestos fibres.

Three of the mass measurements shown in Table 9.2 — 460.7; 6,400; and 104 ng/m^3 — are considerably higher than the rest. Two of these high readings are associated with high fibre counts, while one, 460.7 ng/m^3, is not. An explanation for the lack of correlation in this one case is that a large asbestos fibre or fibre bundle would be counted as a single fibre, but might have considerable mass.

The Ontario data presented in Tables 9.1 and 9.2 are consistent with other studies suggesting that maintenance and inspection activities in the vicinity of asbestos insulation can substantially increase asbestos fibre concentrations in the air.

D.3 Conversion of Transmission Electron Microscopy Results into Optical Fibre Count Equivalents

Earlier in this chapter we identified the three methods of measuring the concentration of asbestos fibres in the air of buildings. These are: measurement of the number of fibres per cubic centimetre of air determined by the optical microscope; measurement of the number of asbestos fibres per cubic centimetre of air determined by the TEM; and measurement of the mass of asbestos fibres in the air by the TEM and expressed as nanograms per cubic metre. Epidemiological studies that relate asbestos exposure to human health express the asbestos exposure in terms of f/cc measured by the optical microscope, or in terms of particle counts, but rarely in terms of mass measurement. In order to assess the possible health effects of the asbestos exposure of building occupants, it would be useful to express that exposure in f/cc as measured by the optical microscope. Yet it has been demonstrated that the optical microscope cannot be used reliably to measure asbestos fibre concentrations in building air. Thus, we must start with TEM measurements and attempt to estimate what their equivalent optical fibre counts would be. We will first consider TEM fibre counts and then TEM mass measurements.

There are two sources of divergence between a TEM count of asbestos fibres longer than 5 microns and the fibre count that would be observed by an optical microscope (PCM) using the standard workplace measuring technique, even when both relate to atmospheres with only asbestos fibres. First, the preparation of a TEM specimen is different from that of an optical microscope slide. Fibre bundles may be broken up into individual fibres in the indirect preparation of TEM specimens, or fibres may be lost. Specimens may become contaminated by asbestos in the laboratory. Current data are insufficient to indicate whether the net effect of these differences in sample preparation will increase or decrease a TEM fibre count relative to a PCM fibre count. Second, the TEM can detect fibres much finer than the thinnest fibres visible under the PCM. Dr. Eric J. Chatfield testified before this Commission that the PCM detects only between 2 and 25% of the fibres longer than 5 microns which would be detected by TEM. Thus, for a given air sample, the optical fibre count might be only 2 to 25% as high as a TEM fibre count.[41] In a subsequent study of asbestos fibres in the Stockholm subway, Chatfield found that of the fibres longer than 5 microns, one-third were sufficiently thick to be visible in an optical microscope.[42] Here the major source of asbestos fibres was thought to be the brake linings of the subway cars, rather than asbestos insulation. Outdoor samples, or samples of indoor air in undisturbed buildings, contain so few asbestos fibres longer than 5 microns that it is not possible to analyze the distribution of fibre thickness and thus to determine reliably the proportion of fibres detected by the TEM that could be seen by an optical microscope. While the actual numbers in particular cases could be as low as 2% or as high as 50%, we conclude that 10% is the best single number to represent this proportion. This implies that a TEM count of fibres longer than 5 microns should be divided by 10, on average, to determine the equivalent optical fibre count.

Pinchin's data in Table 9.2 contain a set of optical fibre counts and TEM counts of fibres longer than 5 microns. The optical counts may be inflated by non-asbestos fibres. The ratio of TEM fibres to optical fibres ranges from a high of 70 to a low of 0.06, with an average of about 17. The variations in these ratios arise from differences in analytical methods, non-asbestos fibres in the PCM data, and the inability of the optical microscope to detect very thin fibres. We interpret them as providing some support for adopting as a rough guideline the assumption that a TEM count of fibres longer than 5 microns should be divided by 10 to estimate the count of fibres that would be detected by PCM. They also show the huge variability in the relationship between the TEM and PCM fibre counts.

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[41] RCA Transcript, Evidence of Dr. Eric J. Chatfield, 9 July 1981, Volume no. 18, p. 140.

[42] Personal communication between Dr. Eric J. Chatfield, Ontario Research Foundation and Royal Commission on Asbestos Staff, 27 June 1983.

Other researchers using the TEM report only the total mass of asbestos and not a fibre count. Can these mass measurements be converted to an equivalent optical fibre count? If every asbestos fibre were the same size (and thus mass), then any mass measurement would represent a specific number of fibres. However, asbestos fibres are of widely varying sizes. Still, if all air samples contained the same proportion of fibres of varying sizes, then it should be possible to determine an average relationship between the number of fibres of asbestos and the mass of those fibres. If this average relationship could be determined, it could be used to convert mass measurements to fibre counts.

Unfortunately, we cannot be confident that the distribution of asbestos fibre sizes is the same in all samples analyzed. The size distribution of fibres will depend upon the type of asbestos involved, the product it is in, and the process generating the dust. Manufacturing may generate different fibre size distributions than the spraying of building insulation or disturbing building insulation. Even in buildings, different types of insulation, or different types of disturbance to that insulation, may release fibres of different sizes and therefore with different fibre to mass ratios. In order to reduce the variability of fibre size distributions, we will concentrate on the measurement of asbestos mass and fibre levels generated by insulation in buildings.

Another problem arises because there is no standard method for making and analyzing mass measurements. Each researcher has developed his own technique for collecting samples and performing the analytical work in the laboratory. Accordingly, the mass measurements made by different researchers are not necessarily comparable. One could hardly expect incomparable mass measurements to be converted to comparable fibre counts.

The conversion between optically visible asbestos fibres longer than 5 microns and asbestos mass is generally expressed as the number of fibres per nanogram of asbestos (f/ng). This means that 1 nanogram of asbestos collected from the air would on average contain the specified number of optically visible fibres. However, measurements of asbestos concentration in the air are expressed in fibres per cubic centimetre and in nanograms per cubic metre. One cubic metre contains 1 million cubic centimetres and therefore 1 million times as many nanograms of asbestos as 1 cubic centimetre. This may be demonstrated by the following example: Consider a conversion of 20 fibres longer than 5 microns measured optically per nano-

gram. If we measure 1 f/cc optically, there will be 1/20 nanogram per cubic centimetre (ng/cc). However, since there are 1 million cubic centimetres per cubic metre, there will be (1 million x 1/20) 50,000 ng/m^3 of asbestos in the air. Thus, 20 f/ng implies that 1 f/cc equals 50,000 ng/m^3. Alternatively, if 30 fibres weigh 1 nanogram, then 1 f/cc equals 33,000 ng/m^3.

Recognizing the limitations discussed above on our ability to convert mass measurements to fibre counts, let us consider the evidence in an attempt to arrive at a conversion factor. Table 9.3 shows the conversions that have been used and derived in other studies. The U.S. EPA reviewed the available evidence and concluded that it was most likely that in building atmospheres 30 fibres of asbestos weigh 1 nanogram, and that 1 nanogram would in any event contain no less than 20 fibres. Dr. William J. Nicholson, as well, stated in his testimony before this Commission that 30 fibres could be regarded as equivalent in weight to 1 nanogram of asbestos.[43] Sebastien, who has conducted extensive mass measurements in buildings, has quoted other studies on the proposition that 2 f/cc measured optically are approximately equal to 100,000 ng/m^3 based on TEM analysis.[44] This conversion means that 1 nanogram of asbestos contains 20 fibres.

Pinchin relied upon electron microscopy performed by Chatfield at the Ontario Research Foundation to measure both the number of fibres greater than 5 microns and the mass of all asbestos fibres. In samples taken from 19 buildings, Pinchin reported data which imply an average of 592 f/ng of asbestos.[45] Analyzing the buildings with amosite separately from those with chrysotile yields 808 f/ng for amosite and 43 f/ng for chrysotile. If the TEM can detect 10 times as many fibres as the PCM, a conversion factor of 59.2 f/ng (optical/electron microscope) is implied by the average of 592 f/ng (electron/electron microscope). Note, however, that when Pinchin used optical measurements alongside TEM mass measurements, the total of his optical fibre counts and the total of his TEM mass measurements implied a conversion factor of 102 f/ng. (See Table 9.2.)

The studies listed at the bottom of Table 9.3 examine the relation of mass to fibre counts in manufacturing workplaces. While these show some consistency with the building data, we do not rely on them for drawing conclusions about building atmospheres.

As the numbers of Table 9.3 show, there is an enormous range in possible conversion factors. Furthermore, there are very few original studies

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[43] RCA Transcript, Evidence of Dr. William J. Nicholson, 29 June 1981, Volume no. 14, p. 100.

[44] Sebastien et al., *Measurement of Asbestos Air Pollution Inside Buildings Sprayed with Asbestos*, p. 26.

[45] Pinchin, *Asbestos in Buildings*, Table II, p. 6.7.

Table 9.3

Relationship Between Fibre Counts and Mass Measurement

Study	Location	f/ng* EM/EM	f/ng* OM/EM
Studies in Buildings			
U.S. EPA**[a]	Buildings		30
Sebastien**[b]	Buildings		20
Pinchin[c]	Buildings	592	
	Buildings		102
Studies in Factories***			
Dement and Harris[d]	Pipe manufacturing (estimated)	76-134	
Winer and Cossett[e]			9.1 36.0
Rohl et al.[f]	Brake maintenance (6 samples)		770
	Brake grinding (2 samples)		62
Bruckman and Rubino**[g]	Not specified		20

Notes: *Fibres counted by electron microscope (EM) or optical microscope (OM). Mass measured by electron microscope (EM).

**Not calculated directly from empirical work, but determined on the basis of other studies.

***These studies are not directly relevant to the conversion of building measurements since they are based on industrial dust measurements.

Table 9.3 (continued)

Relationship Between Fibre Counts and Mass Measurement

SOURCES: Adapted from:

[a] U.S., Environmental Protection Agency, Office of Toxic Substances, *Support Document for Final Rule on Friable Asbestos-Containing Materials in School Buildings: Health Effects and Magnitude of Exposure* (Washington, D.C.: U.S. Environmental Protection Agency, January 1982), p. 95.

[b] Patrick Sebastien et al., *Measurement of Asbestos Air Pollution Inside Buildings Sprayed with Asbestos*, translation of document prepared for the Government of France, Ministry of Health and Ministry of the Quality of Life Environment, 1977, EPA-560/13/80-026 (Washington, D.C.: U.S. Environmental Protection Agency, August 1980), p. 26.

[c] Results calculated from data presented in Donald J. Pinchin, *Asbestos in Buildings*, Royal Commission on Asbestos Study Series, no. 8 (Toronto: Royal Commission on Asbestos, 1982), Table II, p. 6.7.

[d] John M. Dement and Robert L. Harris, Jr., *Estimates of Pulmonary and Gastrointestinal Deposition for Occupational Fiber Exposures*, U.S. Department of Health, Education and Welfare, National Institute for Occupational Safety and Health, Publication no. 79-135 (Cincinnati, Ohio: DHEW/NIOSH, April 1979), pp. 56–58.

[e] Winer and Cossette, 1979, cited in Source d, Table 25, p. 94.

[f] Arthur N. Rohl et al., "Asbestos Exposure During Brake Lining Maintenance and Repair," *Environmental Research* 12 (1976): 125. Data calculated by averaging the data in Table 5, p. 125.

[g] Leonard Bruckman and Robert A. Rubino, "Asbestos: Rationale Behind a Proposed Air Quality Standard," *Journal of the Air Pollution Control Association* 25:12 (December 1975): 1209.

based on empirical fibre counting in buildings. This means that a single conversion factor must be subject to considerable uncertainty. We will use the 30 f/ng conversion that has been suggested by the U.S. EPA, recognizing that in individual measurements the actual relationship might be reflected by a higher or lower number. This conversion suggests more fibres per nanogram than Sebastien's suggestion of 20, but fewer than Pinchin's 102.

As noted above, if there are 30 fibres longer than 5 microns measured optically per nanogram of asbestos in buildings, then a fibre concentration of 1 f/cc would imply a mass density of 33,000 ng/m^3.

The most conservative conversion factor between optical fibres and TEM mass would be the highest factor based on building data, which is 102 f/ng, calculated from Pinchin. Using this conservative conversion factor yields the highest estimate of fibre exposure, and thus the highest estimate of health risks. This is almost certainly too high, since the optical measurements presumably contain some non-asbestos fibres. We will rely on the conversion factor of 30 f/ng because we believe that it is the best single estimate of the true relationship. We will present some calculations using the conservative conversion factor to show the outer limit of possible risks, although we believe it unlikely to represent the actual risks.

Although we have adopted a conversion factor to allow us to express mass measurements as equivalent optical fibre counts, we still have only an approximate index of health risk because we do not know for certain which fibre sizes are most hazardous, and how well *any* of the measurement methods correlate with the actual hazard.

D.4 Conclusion

The data presented above suggest that most asbestos fibre exposures in buildings would be equivalent to concentrations of 0.001 f/cc when measured by optical methods. The highest exposures in buildings would be equivalent to less than 0.01 f/cc. We will review the exposure data in the context of these two numbers to test their validity as benchmarks for describing the exposure of building occupants to asbestos fibre concentrations.

We summarize the available data in Table 9.4, with mass measurements converted to optical fibre counts, recognizing the uncertainty that attends any attempt at conversion. Here we use the conversion of 30 fibres measured optically per nanogram of asbestos, which means that 1 f/cc equals 33,000 ng/m^3. The data from Sebastien show that in buildings with sprayed asbestos insulation, the median exposure level is less than 5 ng/m^3, or 0.00015 f/cc, and 90% of all measurements are less than 30 ng/m^3, or

Table 9.4

Range of Fibre Exposures in Buildings with Asbestos

Study	Mass Measurement ng/m^3	Equivalent Optical f/cc, assuming that 1 f/cc optical = 33,000 ng/m^3
Sebastien[a]		
Outdoors		
– arithmetic mean	0.96	0.00003
– 99% less than	7	0.0002
7 asbestos-free buildings		
– arithmetic mean	1.9	0.00006
21 buildings with spray asbestos		
– lowest building average	1	0.00003
– highest building average	70	0.0021
– highest single reading	751	0.023
– 94% of measurements less than	100	0.003
– 50% of measurements less than	5	0.00015
Nicholson[b]		
Outdoors		
– 83% less than	20	0.0006
– 96% less than	50	0.0015
– highest	87	0.0026
2 asbestos-free buildings (12 samples)		
– 92% less than	20	0.0006
– highest	42	0.0013
17 buildings with sprayed asbestos wet-applied (28 samples)		
– 93% less than	20	0.0006
– highest	180	0.0054
dry-applied (53 samples)		
– 53% less than	20	0.0006
– 83% less than	50	0.0015
– 92% less than	100	0.003
– highest	830	0.025
Sawyer and Spooner[c]		
Connecticut office buildings		
– average (measurement)	79	0.0024
– highest	110	0.0033
New York office buildings		
– lowest building average	2.5	0.000075
– highest building average	200	0.006
Buildings with disturbing custodial activity		
– school average*	643	0.019
– apartment**	296	0.0089
U.S. EPA[d]	57-270	0.0017 - 0.0081
Pinchin***[e]		0 - 0.0003

Notes: *Based on two air samples.

**Based on one air sample.

***Based on TEM fibre count, and assuming that the TEM detects 10 times as many fibres over 5 microns in length as the optical microscope.

Table 9.4 (continued)

Range of Fibre Exposures in Buildings with Asbestos

SOURCES: Adapted from:

[a] Patrick Sebastien et al., *Measurement of Asbestos Air Pollution Inside Buildings Sprayed with Asbestos,* translation of document prepared for the Government of France, Ministry of Health and Ministry for the Quality of Life Environment, 1977, EPA-560/13/80-026 (Washington, D.C.: U.S. Environmental Protection Agency, August 1980), p. 14.

[b] William J. Nicholson, Arthur N. Rohl, and Irving Weisman, *Asbestos Contamination of the Air in Public Buildings,* EPA-450/3-76-004 (Research Triangle Park, N.C.: U.S. Environmental Protection Agency, October 1975), Table 5, p. 25; Table 6, p. 26.

[c] Robert N. Sawyer and Charles M. Spooner, *Sprayed Asbestos-Containing Materials in Buildings: A Guidance Document, Part 2,* prepared for the U.S. Environmental Protection Agency, March 1978, EPA-450/2-78-014 (Washington, D.C.: U.S. Environmental Protection Agency, March 1979), Table I-2-1, p. I-2-9.

[d] U.S., Environmental Protection Agency, Office of Toxic Substances, *Support Document for Final Rule on Friable Asbestos-Containing Materials in School Buildings: Health Effects and Magnitude of Exposure* (Washington, D.C.: U.S. Environmental Protection Agency, January 1982), p. 72.

[e] Donald J. Pinchin, *Asbestos in Buildings,* Royal Commission on Asbestos Study Series, no. 8 (Toronto: Royal Commission on Asbestos, 1982), Table I, p. 7.5; Table II, p. 7.8; Table III, p. 7.12.

0.001 f/cc. We conclude that most buildings with sprayed asbestos insulation expose occupants to less than 0.001 f/cc. The worst building averaged 70 ng/m^3, or 0.0021 f/cc, and 94% of all measurements in all buildings were less than 100 ng/m^3, or 0.003 f/cc. In addition, we conclude that the worst building exposures are considerably less than 0.01 f/cc. Even using the conservative factor of 102 f/ng, the median exposure is still less than 0.001 f/cc, and the worst about 0.007 f/cc.

The Nicholson data show that buildings with wet-applied sprayed asbestos do not expose occupants to fibre levels above those of buildings which do not contain asbestos insulation, or above those of the outdoor air. Buildings with dry-applied sprayed asbestos expose occupants on average to less than 20 ng/m^3, or 0.0006 f/cc. The data show that 92% of all measurements are below 100 ng/m^3, or 0.003 f/cc. Thus, again we can conclude that the average building containing sprayed asbestos insulation exposes occupants to less than 0.001 f/cc, while the worst may expose occupants to considerably less than 0.01 f/cc. Even using the conservative conversion factor, the average building exposes occupants to about 0.002 f/cc and the worst building to less than 0.01 f/cc.

The data from Sawyer and Spooner are less complete and therefore more difficult to interpret. Still, the worst building exposes occupants to 200 ng/m^3, or 0.006 f/cc (or 0.02 f/cc using the conservative conversion), while the average building is clearly far below this.

Turning to the data from Pinchin contained in Table 9.1 above, and dividing his TEM fibre counts by 10 to determine the equivalent optical fibre count, we have a range of 0 to 0.0003 f/cc for undisturbed buildings, well below the 0.001 level. Finally, the U.S. EPA concluded that schools that presented the highest exposure risk for occupants might expose occupants to between 57 and 270 ng/m^3 or, using the conversion factor of 30, expose occupants to between 0.0017 and 0.0081 f/cc.[46] The conservative conversion factor of 102 would yield a range of 0.006 and 0.028 f/cc. The EPA relied primarily on the Sebastien data summarized above and seems to have used the highest measurements as if they were averages, not maxima.

In conclusion, studies of buildings which appear to present substantial asbestos problems show a range of fibre concentrations. Converting these to optical fibre count equivalents so that we can compare these fibre levels with the health effects from occupational exposure, we conclude that the majority of exposures of building occupants in buildings with substantial amounts of friable asbestos would be to fibre levels less than 0.001 f/cc, with a few single readings as high as 0.01 f/cc representing the highest likely exposure.

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[46] U.S., Environmental Protection Agency, *Support Document for Final Rule. . .*, p. 72.

How many hours per week do people spend in the types of asbestos-containing buildings discussed above? Such buildings include schools, office buildings, factories, and warehouses, but not, in general, houses or apartment buildings. Most people are in such buildings for a normal workday or less. It follows that the number of hours per week of exposure of building occupants would tend not to exceed the number of hours per week of potential exposure experienced by industrial workers.

For how many years of his life is the average building occupant exposed? Many people will be in buildings which might contain asbestos during their school and working years, which could total up to 60 years. We will conclude in Chapter 10, however, that less than 10% of schools, offices, factories, and warehouses contain sprayed asbestos insulation. Thus, the average person who spends 60 years in these types of buildings would spend on average 1/10 of those years, or 6 years, in buildings with sprayed asbestos insulation. To be conservative, we will assume an average exposure of 10 years, although this is certainly higher than the average. We recognize, however, that some individuals will spend more than 10 years in buildings containing sprayed asbestos insulation.

It is clear from the above data that the exposure of building occupants to asbestos fibres is enormously less than the exposure allowed to industrial workers under the recently adopted Ontario asbestos control limits of 1 f/cc for chrysotile, 0.5 f/cc for amosite, and 0.2 f/cc for crocidolite. The 0.001 f/cc exposure which we conclude to be the highest estimate of the average exposure in such buildings is 1/1,000 of the maximum worker exposure under the control limit for chrysotile. Many of the buildings that contain sprayed asbestos contain a mixture of chrysotile and amosite. The control limit for amosite is 0.5 f/cc. The highest average exposure is 1/500 this amount. In Chapter 7 we recommend that workers not be exposed to amosite at greater concentrations than would occur with a control limit of 0.1 f/cc. The highest average exposure is 1/100 this amount. Taking 0.01 f/cc as the highest likely occupant exposure, this is 1/100 the exposure of the maximum worker exposure under the new occupational control limit for chrysotile. This is 1/50 the maximum worker exposure under the current Ontario amosite control limit and 1/10 the maximum worker exposure under our recommended amosite control limit. Most exposures are less than 20 ng/m^3 which is 1/10,000 of the allowable exposure of 0.2 milligram (200,000 ng/m^3) allowed under the *Quebec Mining Act*.[47] Asbestos insulation workers and manufacturing workers during the 1950s were exposed to between 1,000 and 10,000 times the asbestos concentrations that current building occupants might experience. One cannot escape the conclusion that the exposure of general building occupants to asbestos fibre concentrations in the air is negligible under most conditions; the conditions under which those exposures are not negligible are readily identifiable.

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[47] *Quebec Mining Act*, R.S.Q. 1977, c. M-13.

The data from Pinchin (Table 9.2 in this chapter), Sawyer (Table 9.4 in this chapter), and others show which conditions can cause elevated fibre levels for building occupants. One such condition is the occurrence of custodial or maintenance work that disturbs friable asbestos-containing insulation. Another condition is the deterioration of insulation so that it falls onto building surfaces and can be disturbed by normal building activity. We conclude that building occupants are not subject to asbestos fibre exposure in excess of a very small fraction of the recently adopted Ontario workplace control limits, except where: (i) the occupant is in the immediate vicinity of work that disturbs friable asbestos-containing insulation; (ii) the occupant is within the range of air circulation of work that disturbs friable asbestos-containing insulation; or (iii) significant quantities of friable asbestos-containing insulation have fallen onto building surfaces and are being disturbed.

We recognize that there are limited data available on the exposure levels of building workers and occupants when maintenance or custodial work is performed on buildings. We anticipate that the implementation of Recommendation 10.22 in the next chapter will shed light on the exposures encountered in the immediate vicinity of building work.

E. Risk Assessment for Building Occupants

If one has developed a model of the dose-response relationship between asbestos fibre exposure and asbestos-related disease, one can use observed exposures to estimate the possible disease that may result from those exposures. This methodology was used in Chapter 7 to estimate the risk to workers caused by different levels of occupational exposure. The risk to building occupants may be similarly estimated, subject to the same high levels of uncertainty in the results. Because there are no reliable epidemiological studies of the health effects of exposures to very low asbestos fibre levels, any risk assessment must involve extrapolation of other data which introduces great uncertainty about the result. We assume a linear dose-response relationship, which, if anything, may overestimate disease.

The U.S. EPA analyzed the health risks to building occupants from exposure to asbestos in a paper first released in 1980 and then released in revised form in 1982. The EPA paper concluded that school buildings containing friable asbestos might expose occupants to airborne asbestos concentrations between 57 ng/m^3 and 270 ng/m^3, and that because of deterioration of the asbestos in the future, this exposure might rise to 500 ng/m^3.[48] Custodial and maintenance workers could experience greater exposures because of their work. These exposure estimates were based primarily on the work of Sebastien in France and on only those buildings in his studies with high asbestos levels. We believe that the EPA conclusions overestimated the average exposure of occupants of buildings containing sprayed asbestos insulation, in part because of a selective use of the highest exposures reported by Sebastien. Of the 21 buildings studied by Sebastien, the EPA analyzed only 8, those with the highest airborne asbestos fibre levels of the 21.

The 1982 EPA paper, unlike the 1980 version, did not mention its selective use of data reported by Sebastien. Nor did it mention that in 9 buildings containing sprayed asbestos insulation the maximum asbestos fibre concentration found by Sebastien was 7 ng/m^3 or less; by Sebastien's definition, not "abnormally high." (See the summary of the Sebastien data in Table 9.4.) We find that this selective use of Sebastien's data in the EPA paper resulted in an overestimation of the average exposure of occupants of buildings containing sprayed asbestos insulation.

The EPA study compared the exposure of building occupants to the exposure of building insulation workers who were exposed to the same types of fibres. The average working exposure of insulation workers, as calculated by the EPA, ranged from 3 to 15 f/cc, with an average estimated exposure of 9 f/cc.[49] The study determined that 30 fibres measured optically are equivalent to 1 ng measured by the TEM.[50] Insulation workers thus received a lifetime exposure of between 2 million and 10 million nanograms per cubic metre-years (ng/m^3-yrs). A student who spends 6 years in a school will be exposed, multiplying the exposure of 57 or 270 ng/m^3 by 6, to between 342 and 1,620 ng/m^3-yrs of asbestos while at that school. The low exposure of the school occupant is 0.00017 times the low exposure of the insulation worker, while the high exposure of the school occupant is 0.00016 times the high exposure of the insulation worker.

While the 1980 draft version of the EPA study proceeded to estimate the amount of disease that might be caused by such exposure, the final document contained no disease estimate. Both the methodology and the numbers were changed somewhat between the draft and the final version, so that it is not possible to produce a disease estimate directly from the final document. However, assuming a linear dose-response relationship for asbestos-related cancers, the data would imply that school occupants might experience about 0.00017 times the disease encountered by the insulation workers studied by Hammond, Selikoff, and Seidman.[51] The 1980 EPA report attributed to Hammond's study 716 excess cancer deaths among 12,051 workers exposed, or 59 deaths per thousand.[52] Multiplying this by 0.00017 yields 0.01 excess deaths per thousand school occupants. This equals 10 deaths per million occupants. While these numbers are subject to considerable uncertainty, they do not suggest a significant risk of asbestos-related disease for building occupants.

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[48] U.S., Environmental Protection Agency, *Support Document for Final Rule. . .* , p. 72.

[49] Ibid., p. 91.

[50] Ibid., p. 94.

Julian Peto has performed an independent assessment of the risk of cancer for school occupants. He assumed that the U.S. insulation workers were exposed to an average of 30 f/cc for 20 years and extrapolated the risks to building occupants from the insulation worker experience, since the *type* of fibre exposure was the same. From his results, it can be calculated that the combined risk of death from asbestos-related lung cancer and mesothelioma for a student exposed for 6 years, from age 12 to age 18, to 0.001 f/cc would be 0.0063 per thousand exposed persons. A staff member exposed for 10 years from age 30 to 0.001 f/cc would face a nearly identical combined risk of 0.0061 per thousand exposed persons. Eighty percent or more of these deaths would occur after age 60.[53] These risks are equal to 6.3 or 6.1 deaths per million exposed persons.

The U.K. Advisory Committee on Asbestos also estimated possible health risks to building occupants. It posited a range of fibre exposures because of the difficulties of both measuring fibre levels in buildings and converting mass measurements to fibre counts. Using the Advisory Committee's median estimate, buildings expose occupants to 0.005 f/cc for 50 years; the cumulative dose is 0.25 fibres per cubic centimetre-years (f/cc-yrs); and the excess lung cancer mortality among building occupants might range from 5 to 170 deaths per million births.[54] The Advisory Committee concluded: ". . . unless contaminated buildings are very much commoner than seems likely no appreciable mortality from lung cancer can be associated with any degree of contamination by chrysotile likely to be encountered in the U.K. in the ambient air or in buildings not under active construction or repair."[55] However, the Advisory Committee emphasized the need for further information about asbestos levels in buildings.

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[51] E. Cuyler Hammond, Irving J. Selikoff, and Herbert Seidman, "Asbestos Exposure, Cigarette Smoking and Death Rates," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 473–490.

[52] U.S., Environmental Protection Agency, *Support Document for Proposed Rule on Friable Asbestos-Containing Materials in School Buildings: Health Effects and Magnitude of Exposure*, EPA 560/12-80-003 (Washington, D.C.: U.S. Environmental Protection Agency, October 1980), Table 17, p. 80.

[53] Julian Peto, "An Alternative Approach for the Risk Assessment of Asbestos in Schools," report to the U.S. Environmental Protection Agency, 6 April 1981, p. 14. (Mimeographed.) Mr. Peto has subsequently stated that the 30 f/cc exposure used in this paper was for illustrative purposes only, and that the Nicholson exposure estimate of 15 f/cc should be used. This change would raise Peto's risk estimates, perhaps to about 12 per million. Letter and attachments from Mr. Julian Peto, Imperial Cancer Research Fund, University of Oxford, Oxford, England to the Royal Commission on Asbestos, 21 September 1983.

[54] U.K., Advisory Committee on Asbestos, *Asbestos — Volume I: Final Report of the Advisory Committee* (Simpson Report), William J. Simpson, Chairman (London: Her Majesty's Stationery Office, 1979), Table 22, p. 62.

The Ontario studies performed for this Commission reinforce the belief that the asbestos exposure of building occupants is low. In the previous section we conclude that the majority of exposures of building occupants would be below 0.001 f/cc with a few exposures as high as 0.01 f/cc, in buildings with friable asbestos insulation. Using the data from the risk assessment for exposed workers to evaluate risks to building occupants, if a contaminated building exposed the occupants to 0.001 f/cc more than the ambient air, the total mortality for a 10-year exposure might be approximately 0.001 times the mortality for a 1 f/cc exposure.

Let us now focus on the analysis of the study by Selikoff, Hammond, and Seidman, since this study examined insulation workers exposed to the types of fibres present in buildings.[56] In the Appendix to Chapter 7 of this Report, the Daniels and Roberts models, using the Selikoff data, yield mortality projections of 20 deaths per million persons exposed for 10 years, which is within the range found by the U.K. Advisory Committee. If we consider the regular occupant of a building area with the highest asbestos fibre levels, estimated in the preceding section as 0.01 f/cc, the risk of death based on the Selikoff data would be 200 per million such exposed persons. We believe that regular exposures of building occupants to fibre levels as high as 0.01 f/cc are unlikely. These estimates are uncertain both in the estimated exposures and in the estimated health effects of the exposures.

Studies of risks to building occupants have indicated a wide range of uncertainty as to the magnitude of those risks. In Chapter 7 we discuss the risks faced by workers, asking whether those risks are significant in comparison to other risks faced by workers. We now ask whether the risks faced by building occupants as a result of exposure to these asbestos fibre levels are significant in comparison to other risks faced by building occupants. In answering this question, we must identify other risks to be used as a basis for comparison.

Table 9.5 summarizes risks of death by various causes faced by Canadians, expressed in deaths per year per 100,000 population. The top part of the table shows fatality rates from accidents for Canadians in the year 1980 per 100,000 population per year. Motor vehicle traffic accidents caused 22.5 deaths per 100,000 population. The risk of death from accidental falls was 7.7 per 100,000. The risk of death from fire and from poisoning was 3.1 and 1.9 respectively per 100,000. The major diseases causing fatalities are shown in the bottom half of Table 9.5. Cardiovascular disease caused 337.2 deaths per 100,000 and cancer caused 165.3; while pneumonia caused only 19.7 and diabetes caused 12.0.

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[55] Ibid., paragraph 132, p. 63.

[56] Irving J. Selikoff, E. Cuyler Hammond, and Herbert Seidman, "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," *Annals of the New York Academy of Sciences* 330 (14 December 1979): 91-116.

To compare the risk from asbestos exposure to these risks, a conversion must be made from the type of data presented by the U.K. Advisory Committee. The U.K. Advisory Committee data estimated the number of deaths per million births. To convert this to deaths per 100,000 population per year we need a population model. We will use a very simple model to produce order-of-magnitude figures for comparison purposes. Assume a state in which 1 million people are born every year, each of them living to age 70. With 1 million persons in each one-year age group, the number of deaths in one year is the same as the number of deaths experienced by a single group of 1 million persons over their 70-year lives. In this state, the population would be 70 million persons, which is 700 x 100,000 persons. Thus, a death rate per million live births or per million exposed persons must be divided by 700 to produce deaths per 100,000 population. The U.K. Advisory Committee estimated 5 to 170 deaths per million live births, which would represent an average of 5 to 170 deaths caused by asbestos exposure per year in this hypothetical state. Dividing this death rate by 700 yields 0.007 to 0.24 deaths per 100,000 population. This is shown at the bottom of Table 9.5.

Above we estimate that there might be 20 deaths if 1 million persons were exposed to building insulation for 10 years at 0.001 f/cc. In our hypothetical state this would cause 20 deaths per year with a population of 70 million — or 0.029 deaths per year per 100,000 population. This is shown at the bottom of Table 9.5. The unlikely exposure to 0.01 f/cc implies a risk 10 times greater than that just calculated, or 0.29 deaths per 100,000.

The asbestos risks shown in Table 9.5 are, if anything, overstated in comparison to the risks of death from other causes. The asbestos risks assume that every member of the population is exposed to asbestos in buildings for either 10 years, when we use Daniels and Roberts, or 50 years, when we use the U.K. Advisory Committee. However, if only 10% of buildings contain asbestos, then on average only a small fraction of the population will be exposed to asbestos in buildings. It would therefore be incorrect to multiply the building risk represented in Table 9.5 by the ratio between the Canadian population and 100,000 in determining the total risk to all Canadians.

The person who works for 10 years in a building with 0.001 f/cc of asbestos might face a risk of death from that 10-year exposure of 20 per

Table 9.5

Risks of Death by Cause — 1980
(Death rate per 100,000 population/year)

Cause of Fatality	
Accidents[a]	
Motor vehicles (traffic)	22.5
Falls	7.7
Drowning	3.4
Miscellaneous	2.9
Fire	3.1
Poisoning	1.9
Suffocation	2.2
Aircraft	0.7
Motor vehicles (non-traffic)	0.6
Firearms	0.3
Disease[b]	
Cardiovascular diseases	337.2
Cancer	165.3
Pneumonia	19.7
Diabetes	12.0
Asbestos Disease from Building Exposure	
U.K. Advisory Committee on Asbestos (50 years)[c]	0.007-0.24
Royal Commission on Asbestos Estimate (10 years)	0.029

SOURCES: [a] Canada Safety Council, *Accident Fatalities, Canada 1980* (Ottawa: The Council, 1982), Table 2, p. 5.

[b] Ibid., Table 19, p. 24.

[c] Adapted from: U.K., Advisory Committee on Asbestos, *Asbestos — Volume 1: Final Report of the Advisory Committee* (London: Her Majesty's Stationery Office, 1979), Table 22, p. 62.

million. If the same person drove 10 miles to and from the building 250 days per year for 10 years, he would drive 25,000 miles. Since the number of fatalities per vehicle-mile travelled in Canada is approximately 45 per billion, the risk of death from commuting is 1,125 per million.[57] In short, the drive is over 50 times as dangerous as the building occupancy.

Are the risks of asbestos exposure to building occupants shown in Table 9.5 significant? Because they are orders of magnitude below the other risks faced by the general population, we conclude that these risks are not significant. While the health risk from asbestos in buildings may not be exactly zero, it is far below risks faced every day by Canadians in their ordinary lives. The risk to occupants from asbestos in buildings is a small fraction of the risks faced by workers exposed to asbestos under the 1 f/cc control limit for chrysotile. It is less than 1/50 as great as the risk of commuting by car to and from those buildings. In concluding that this risk is insignificant, we conclude that the risk does not present a public health problem. While asbestos has caused serious health problems for workers and may present a problem for building maintenance, renovation, construction, and demolition workers, we conclude that it does not pose a significant problem for the general occupants of a building, except in the three situations outlined in Section D of this chapter, namely: (i) the occupant is in the immediate vicinity of work that disturbs friable asbestos-containing insulation; (ii) the occupant is within the range of air circulation of work that disturbs friable asbestos-containing insulation; or (iii) significant quantities of friable asbestos-containing insulation have fallen onto building surfaces and are being disturbed.

While we have emphasized the uncertainties inherent in the risk assessment performed here, we can also indicate the boundaries of that uncertainty. First, the estimates of the fibre exposure of building occupants is subject to considerable uncertainty because of the absence of a standardized methodology for measuring fibre levels. Still, all the available exposure estimates indicate that few asbestos-containing buildings expose their occupants to as much as the equivalent of 0.01 f/cc of asbestos measured optically, and the best exposure estimate is less than the equivalent of 0.001 f/cc measured optically. This exposure is 100 to 1,000 times lower than the current workplace control limit for chrysotile. It is 1,000 to 10,000 times lower than the average exposure of insulation workers in the past. While the range of uncertainty in these exposures is large, all are at extremely low levels.

Second, there is not a well-established dose-response relationship between low exposures to asbestos fibres and asbestos-related diseases. We conclude in Chapter 5 that a linear relationship is both reasonable and prudent, in that it is unlikely that the true relationship would cause more disease than is predicted by a linear relationship.

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[57] Transport Canada, *1980 Canadian Motor Vehicle Traffic Accident Statistics*, TP 3322 (Ottawa: Transport Canada, 1980).

What would it cost per person-year per fibre to reduce this exposure? Assume an office building that would be occupied by 1,000 workers for a period of 10 years. The average Toronto office building has 25 square metres of floor space for every worker.[58] Thus, a building housing 1,000 workers would occupy 25,000 square metres. We will assume, based on Pinchin, and given that prices for removal work have been dropping, that the cost of removing and replacing sprayed insulation is $50 per square metre.[59]

If the building is occupied, the occupants must be moved out and back, which Pinchin estimated might cost $15 to $30 per square metre for office employees.[60] We will use $20 per square metre. Alternate space must be found for the duration of the control work, and we will assume that this costs $20 per square metre for one month. The sum of these removal, moving, and space costs is $90 per square metre.

All of this cost must not be attributed to the immediate control programme, since we are recommending that the asbestos would have to be removed before the building could be demolished. (See Chapter 10.) The net cost of present control work is the cost of that work less the cost that would be incurred at demolition. Assume that the building would not be demolished for 20 years. Delaying asbestos removal until just prior to demolition will be less costly than if the building will be re-used, since less care must be exercised to avoid damaging the building and furnishings. We will assume that removal at demolition saves 25%, yielding a cost of $37 per square metre. Since this cost would occur 20 years in the future, we must determine the present value of that amount today, since a lesser sum of money could be set aside today, which, with accumulated real interest, would amount to $37 in real terms in 20 years. Using a 5% real discount rate, the present value today of $37 in 20 years is $11.53. The net cost of asbestos removal in an office building today could therefore be estimated at ($90 - $11.53) about $80 per square metre. If we apply this cost to our hypothetical building, the cost of a removal project would be $1.6 million. Dividing this control cost by an exposure reduction of 0.001 f/cc x 1,000 persons x 10 years yields a marginal cost of $160,000 per person-year per f/cc. This calculation implies that a removal project could reduce fibre levels from 0.001 to 0, and hence overestimates the effectiveness of a removal project, since even buildings without asbestos-containing insulation have background levels of asbestos. If the actual fibre level is 0.01 f/cc, the cost is 1/10 as great per f/cc, or $16,000 per person-year per f/cc.

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[58] Telephone communication between Mr. Peter Tomlinson, Program Manager, Policy Section, Planning and Development Department, City of Toronto and Royal Commission on Asbestos Staff, 4 July 1983.

[59] Pinchin, *Asbestos in Buildings*, p. 8.9.

[60] Ibid., p. 5.21.

In Chapter 6 we show that controlling worker exposures in brake manufacturing to an average exposure of 0.5 f/cc might cost $2,700 per person-year per fibre. Thus, the cost of protecting building occupants at $160,000 per person-year per fibre is enormously greater than the cost estimated for applying controls in the workplace, largely because the building exposures are insignificant.

We estimate above that an exposure of 0.001 f/cc for 10 years might create a risk of premature death of 0.02 per 1,000 exposed persons. Spending $1.6 million to eliminate this risk implies an expenditure of $80 million per possible life saved.

The evaluation of these costs of possible life saving may be compared with estimates of the costs of life saving that might result from other public programmes. If a limited amount of money is to be spent in protecting the health and longevity of Canadians, the greatest improvement in health and longevity will be achieved by spending that money where the cost per life saved is the lowest, so that the amount of life saving per dollar is greatest. Unfortunately, there are very few studies of the costs of life savings from public programmes in Canada. Byer, Bacchus, and Melcher analyzed the safety impact of 12 grade separation projects in Ontario from 1958 through 1976 in which level crossings between highways and railroads were replaced by bridges and underpasses. This study found that the highest cost among these 12 projects was $16 million per estimated life saved, in 1979 Canadian dollars.[61] All values of the study, however, were quite sensitive to several of the assumptions.

In Chapter 7 we discuss a study that found that in the United States the median cost per life saved in various programmes was $64,000 in motor vehicles and highways; $102,000 in medical programmes; $50,000 in consumer protection; $2.6 million in environmental protection; and $12.1 million in occupational health and safety programmes.[62] Not all the programmes analyzed have in fact been adopted by the U.S. government, but these figures probably give a rough estimate of the range of expenditures per life saved experienced in the United States. The estimated cost of possible life saving for building occupants from asbestos control programmes is far above the cost of life saving in other programmes. From the standpoint of occupants, asbestos control in buildings is uneconomic because the risk it removes for occupants is insignificant, and its costs are large.

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[61] Philip H. Byer, Ataur Bacchus, and Rowena Melcher, *Implying the Value of Life From Public Safety Investments*, Research Report, no. 64 (Toronto: University of Toronto-York University Joint Program in Transportation, March 1980), p. 24.

[62] John D. Graham and James W. Vaupel, "Value of a Life: What Difference Does it Make?" *Risk Analysis* 1:1 (March 1981): 91.

Our conclusion that the occupants of buildings are generally not at risk from asbestos fibre exposure, and that asbestos control work may expose workers to substantially elevated asbestos fibre levels if not carefully performed, raises the question whether the programme to remove asbestos from all Ontario schools, involving an expenditure of provincial funds of over $26 million to date, constituted an over-reaction, albeit well intentioned, to public concern vigorously expressed by parents alarmed by perceived health risks to their children. We appreciate that children may be somewhat more at risk from a given fibre exposure than are adults, as discussed in Chapter 5 above. The risk assessment models presented in the Appendix to Chapter 7 predict that children may be exposed to almost twice the risk faced by adults. Even acknowledging that the very young may be more susceptible to asbestos disease, the health risks to children remain insignificant because the level of exposure in asbestos-containing schools has in general been extremely low. The exceptions would be cases where asbestos material was actively disturbed, or where it fell onto building surfaces and was disturbed. It follows that, with these exceptions, the programme for removing asbestos from all asbestos-containing schools was not justified by the health risk posed to students. In some cases, the asbestos control work may have been justified in order to protect building renovation, maintenance, or custodial workers from exposure to asbestos if their work would have disturbed asbestos insulation. (See Chapter 10, Section D.) But neither the scale nor the pace of the school programme was warranted by the risk posed by most asbestos-containing schools to occupants or workers. If anything, the scale and pace of the programme significantly *increased* the risk to some workers directly engaged in control projects. Crash programmes invariably mean that inexperienced contractors and personnel will enter the field. A number of asbestos-control projects in schools were conducted in a manner which, according to the evidence before us, may have generated significant risk for the workers involved. (See Chapter 10, Section D.) There is no question, however, that the crash nature of the asbestos control programme in schools was occasioned by a climate of public apprehension; the actions of school boards and of the Ministry of Education, paralleled in other jurisdictions, show them to be responsive to public demand.

F. Recommendations for Occupant Protection

We conclude that a universal programme of building inspection and control is not warranted to protect building occupants. As Pinchin has noted, this would be quite expensive. In addition, it is unlikely that such a programme could be performed at a high level of competence in a short period of time, given the limited number of individuals who are adequately trained in this work and the length of time required to develop the necessary training and experience. Under the circumstances, only a selective approach to inspection and control is warranted.

In Section D of this chapter we conclude that building occupants are not subject to asbestos fibre exposure in excess of very small fractions of the recently adopted Ontario workplace control limits, except where: (i) the occupant is in the immediate vicinity of work that disturbs friable asbestos-containing insulation; (ii) the occupant is within the range of air circulation of work that disturbs friable asbestos-containing insulation; or (iii) significant quantities of friable asbestos-containing insulation have fallen onto building surfaces and are being disturbed. The recommendations we shall make in Chapter 10 for the protection of workers should also protect building occupants in the situations described in (i) and (ii) above. This leaves situation (iii). When significant quantities of insulation have fallen onto building surfaces and are being disturbed, this should be readily apparent to building occupants and to the person disturbing the fallen insulation. In buildings in which insulation was installed or replaced before 1974, the year when the use of asbestos in insulation ceased in Ontario, a person who observes such fallen insulation should have a right to have it tested at the expense of the building owner to determine whether it contains asbestos. If an occupant and the owner disagree on whether a "significant quantity" of insulation has fallen, the responsible Ministry could be called upon to assess the situation. We accordingly recommend that:

9.5 *Legislation should be developed providing that, if significant quantities of friable insulation have fallen onto building surfaces and are being disturbed, the building owner must have the material tested to determine whether it contains asbestos. This requirement should apply only to insulation installed or replaced before 1974.*

We further recommend that:

9.6 *Legislation should be developed providing that if the test mandated by the circumstances stipulated in Recommendation 9.5 shows that friable asbestos is present, the building owner must enclose, encapsulate, or remove the asbestos-containing material, following the work practices referred to in Recommendation 10.17.*

We consider that a programme of management and custodial control should be instituted as soon as the friable material is found to contain asbestos and should continue until the asbestos-containing material is removed: see Recommendation 10.6. As well, the results of testing under Recommendation 9.5 should be available to government inspectors, building workers, and building occupants: see Recommendation 10.3.

We have refrained from specifying which Ministry should be responsible for the implementation of Recommendations 9.5 and 9.6. The subject matter of these recommendations suggests a role for the Ministry of Health. However, the Ministry of Labour will have expertise in dealing with asbestos in buildings, because the main concerns raised by asbestos in buildings involve workers. We recommend that:

9.7 The Minister of Labour should communicate with the Minister of Health with a view to establishing responsibility with respect to Recommendations 9.5 and 9.6.

G. Asbestos in Private Homes

Friable asbestos has been used in private homes as insulation on cast iron hot water boilers for heating or for domestic hot water and as insulation on heating pipes. This asbestos is usually confined to the basement or to the furnace room in the basement. Any hot water or steam heating system that was installed or re-insulated before the mid-1970s might include asbestos insulation. It seems likely that a large proportion of cast iron hot water boilers currently in use in homes in Ontario would include some asbestos insulation.

The mere presence of such asbestos insulation does not pose a health risk. Only when the covering on the insulation is damaged so that dust can escape, *and* there is nearby activity such as that associated with a laundry, recreation room, or workshop, is there a possibility of asbestos fibre exposure for the building occupants. If the boiler is in a separate room that is never used by building occupants, there should be no risk. Because asbestos in private homes is unlikely to present health risks to building occupants, because the owner is usually the occupant, and because of the difficulties of enforcing regulations upon the large number of private homes in Ontario, it is not appropriate that the duties of Recommendations 9.5 and 9.6 above be applied to such homes. Accordingly, we recommend that:

9.8 Owners of private homes should be exempt from the duties set out in Recommendations 9.5 and 9.6 above.

The risk, if any, presented by asbestos insulation in private homes is not to the building occupant, but to workers who may disturb that asbestos in performing maintenance work on the boilers or pipes. We therefore recommend that:

9.9 The Ministry of Labour should communicate with workers who replace and maintain domestic space-heating boilers to inform them of the risks of working with asbestos and of the appropriate safety precautions for such work.

While asbestos-containing pipe or boiler insulation will be found in many homes, in most cases there will be no risk to building occupants. When a risk to building occupants or workers is suspected, because the insulation is damaged and there is activity nearby, tests should be performed to determine whether or not asbestos is present. If it is present, it can be controlled in place or, if necessary, removed. Because removal necessarily disturbs the asbestos and presents some risk of exposure both to the removal worker and to the occupants of the house, removal is not recommended unless a definite risk is identified. To control the asbestos in place, the homeowner should avoid unnecessary activity which might disturb the asbestos. In addition, workers whose work might disturb the asbestos should be informed that asbestos is present, so that safe work practices can be employed. We recommend that:

9.10 The Ministry of Labour should prepare a brief flyer describing the possible locations of asbestos on pipes and boilers in the home, the proper method of sampling suspect material, and any appropriate precautions should asbestos be found to be present.

This flyer could be supplied to municipalities throughout the province, to be distributed to ratepayers with their property tax bills.

Other asbestos building products which might be found in homes include floor tiles and asbestos-cement sheet. These are discussed in Chapter 10 and present no risk to the occupant of the house in ordinary use. We have investigated the possibility that loose asbestos may have been used as attic insulation in private homes. We find no evidence that asbestos was so used on a commercial scale, although a few homes may contain such insulation, probably installed by the homeowner himself. Apparently asbestos has always been more expensive than competing loose insulation of equivalent insulation capability, so that there was no reason to use asbestos in attics.

Chapter 10 Protecting Workers from Asbestos in Buildings

A. Introduction

In Chapter 9 we conclude that the presence of friable asbestos in buildings does not present a health hazard to building occupants except in special circumstances where work disturbs friable asbestos-containing insulation or where substantial quantities of friable asbestos-containing insulation have fallen onto building surfaces and are being disturbed. In contrast, the same data that show the relative insignificance of most building occupant exposures also show that workers who disturb asbestos-containing insulation may be exposed to fibre levels at or above Ontario control limits. The removal of asbestos-containing insulation generally causes very high dust levels. Enclosing or encapsulating asbestos-containing insulation may cause high dust levels; working above a suspended ceiling which encloses sprayed asbestos may cause high dust levels; and work on a surface covered with asbestos-containing insulation may cause high dust levels. As well, working with new building products which contain asbestos causes elevated fibre levels, not for building occupants, but for the tradesmen who work on the building itself.

This chapter explores the magnitude of the problem of worker exposure to asbestos in buildings and the steps necessary to protect the health of the workers concerned. We consider that these steps should be incorporated in a regulatory instrument which is separate and distinct from the Regulation Respecting Asbestos that was promulgated in 1982. The Regulation Respecting Asbestos was drafted with the manufacturing and mining sectors in mind, imposing numerical asbestos control limits and relying on air monitoring for its enforcement. It does not apply to work on construction projects. The Ministry of Labour intends to regulate asbestos exposure on such projects, which include activities related to removal and demolition, by framing a separate regulation under the *Occupational Health and Safety*

Act. It published an exposure draft of this regulation, entitled Proposed Regulation Respecting Asbestos on Construction Projects and Related Codes (PRRACP), in *The Ontario Gazette* in August 1982 and presented a revised version at a public meeting in January 1983.[1]

The PRRACP relies on regulation by procedure rather than on control limits. However, the scope of the PRRACP is limited to what is defined in section 1, paragraph 23 of the *Occupational Health and Safety Act* as "construction," which includes erection, alteration, repair, dismantling, demolition, and structural maintenance. We endorse regulation by procedure on construction projects so defined, but consider that this approach is likewise appropriate to activities that involve building maintenance and custodial work. Regulation by procedure specifies procedures to protect worker health and safety and provides an immediate means of deciding whether a given work practice is safe.

Building maintenance and custodial work, like construction projects as defined in the Act, cannot be regulated appropriately through the fixed workplace approach of imposing control limits enforced by air monitoring. As we observed in Chapter 9, Section C, the only standardized workplace measuring technique, phase contrast microscopy, cannot distinguish asbestos dust from non-asbestos dust. Since most building work can be expected to raise significant amounts of non-asbestos dust, it is not practicable to regulate this work through reliance on air monitoring. Furthermore, fibre levels can vary tremendously in building or construction work, from time to time and from place to place on the same job. This is because the nature of the work and the material being worked on vary as the job progresses. Even if air monitoring took place every hour of every day, the laboratory analysis might not be completed for a few days, often too late to be useful if the work that caused high exposures has been replaced with some other work.

We, therefore, conclude that regulation by procedure, because of its immediate focus on safe work practices, provides the appropriate means of regulating asbestos exposures that might be encountered in building-related work activities, whether these be on construction projects or in association with building maintenance and custodial work. It follows that the scope of regulation by procedure envisaged by the Ministry's PRRACP should be broadened to extend beyond construction projects to maintenance and custodial work in buildings. Accordingly, the Ministry of Labour should proceed to frame a regulation pursuant to the *Occupational Health and Safety Act* on asbestos on construction projects and in buildings and should amend the existing Regulation Respecting Asbestos so as to specify that its terms do not apply to work activities covered by the new regulation. We therefore recommend that:

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[1] Ontario, Ministry of Labour, Occupational Health and Safety Division, "Proposed Regulation Respecting Asbestos on Construction Projects and Related Codes," presented at a Public Meeting, 17 January 1983, Toronto. (Mimeographed.) This "Proposed Regulation. . ." is a revision of the "Notice of Proposed Regulation: Designated Substance — Asbestos on Construction Projects," *The Ontario Gazette*, vol. 115-33, Saturday, 14 August 1982, pp. 3194-3197.

10.1 The Ministry of Labour should frame a regulation under the *Occupational Health and Safety Act* *respecting asbestos on construction projects and in buildings which includes building custodial and maintenance work. The Ministry should amend the existing Regulation Respecting Asbestos so as to specify that its terms do not apply to work activities encompassed by the new regulation.*

How widespread is the exposure of building workers to elevated asbestos fibre levels? Not every building in Ontario contains friable asbestos. For the most part, asbestos-containing insulation was not applied in Ontario buildings after 1973. Sprayed asbestos-containing insulation is extremely rare in residential buildings of any kind and is most common in non-residential, multi-storey buildings. We estimate that less than 10% of non-residential, multi-storey buildings in Ontario contain sprayed asbestos-containing insulation and less than 20% contain extensive asbestos-containing pipe and boiler insulation.

Even in those buildings which contain friable asbestos insulation, the possibility of a health risk for workers is largely limited to situations where work will disturb this asbestos. As in Chapter 9 we conclude that there is no justification for a universal programme of building inspection to discover the presence of asbestos for the protection of occupants, so here we will conclude that such universal building inspection is not required to protect workers. Even less is a universal programme of asbestos removal called for. What is generally necessary is to test or inspect for the presence of asbestos prior to work that may disturb friable fibrous material. Control work, other than the basic management required as soon as friable asbestos is discovered in a building, becomes necessary only when the presence of asbestos is proved and the work activity to be performed on or near it suggests that there will be a hazard to building workers. Therefore, most asbestos control work will be performed prior to the demolition, major renovation, or major maintenance of a building. When minor renovation or maintenance is contemplated, the application of safe work practices will usually be sufficient to protect workers from excessive exposures.

Identifying situations that may present health risks to workers requires imposing some duties on building owners, contractors, and workers themselves. Building owners, contractors, and workers must be instructed in identifying possibly hazardous situations and in safe practices to be used in those situations. Qualifications must be established for those who inspect buildings for asbestos and for those who supervise and inspect asbestos control work.

The overall thrust of the recommendations in this chapter is to ensure that asbestos-containing material is identified before it is disturbed, and that adequate protective measures are taken when it is disturbed to ensure that workers are not exposed to potentially significant fibre levels. The remaining sections of this chapter will describe an approach to inspecting buildings for asbestos hazards, corrective actions that may be taken to control asbestos, and safe practices for working with asbestos in buildings.

In Chapter 7 of this Report we identify the appropriate control limits for worker exposure to asbestos in fixed workplaces, and in Chapter 8 we devote considerable effort to examining the problems of implementing this control limit. Concern with asbestos in buildings and on construction projects is more recent than concern with asbestos in fixed workplaces. There is much less experience with regulating asbestos in buildings than with regulating asbestos in fixed workplaces and therefore less of a foundation upon which this Commission might build recommendations for effective implementation. We have attempted to be as realistic as possible in our recommendations, recognizing the helpful input received from written submissions and testimony regarding the experience of workers with asbestos on construction projects in this province. We recognize, however, that our treatment of implementation is, for the reasons just stated, less complete in the area of asbestos in buildings than of asbestos in fixed workplaces. We urge that as the Ministry of Labour, workers, and industry develop experience in this regulatory area, careful note be taken of the problems of implementation so that appropriate adjustments may be made.

B. Inspecting Buildings for Asbestos

B.1 Current Programmes of Inspection

In the United Kingdom, the Advisory Committee on Asbestos concluded that the risk to building occupants from asbestos fibre exposure was not sufficient to warrant either removal of all asbestos from existing buildings or a universal programme of asbestos identification in buildings.[2] A large number of building owners in the United Kingdom, in both the private and public sectors, have nonetheless undertaken complete asbestos surveys of their buildings even though this is not required by legislation.[3]

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[2] U.K., Advisory Committee on Asbestos, *Asbestos — Volume I: Final Report of the Advisory Committee* (Simpson Report), William J. Simpson, Chairman (London: Her Majesty's Stationery Office, 1979), paragraph 248, p. 91.

[3] Donald J. Pinchin, *Asbestos in Buildings*, Royal Commission on Asbestos Study Series, no. 8 (Toronto: Royal Commission on Asbestos, 1982), p. 2.2.

In the United States, there has been no mandatory universal programme for inspecting private or public buildings for asbestos. However, in May 1982, the U.S. Environmental Protection Agency (EPA) published in the *Federal Register* a requirement that public, elementary, and secondary schools be inspected for friable asbestos-containing materials. School officials must maintain records of the inspection results, notify employees of the locations of friable asbestos materials, and provide employees with instructions on reducing exposure to asbestos.[4] The rule does not require that any particular control action be taken. In general, building inspection for asbestos in the United States is concentrated in school buildings. Two types of inspection programmes have been carried out in Ontario. In the first type, an organization establishes a programme to inspect all buildings within its jurisdiction that may contain asbestos. In the second type, buildings are inspected only when concern is expressed over particular problems in particular buildings.[5] In his study for this Commission, Dr. Donald J. Pinchin described examples of current inspection programmes in Ontario.[6] The Ministry of Education, which has jurisdiction with respect to schools, has supervised a comprehensive inspection programme. Public Works Canada, the National Capital Commission, and the City of Windsor Utilities Commission have also performed comprehensive inspections of buildings within their purview. On the other hand, the Ontario Ministry of Government Services has only inspected buildings in response to complaints.

All federal departments have completed inspection programmes and are carrying out corrective work.[7] In addition, a number of building owners in the private sector have begun or completed their own inspection programmes. It would seem that most inspection programmes in the private sector follow the approach, adopted by the Ontario Ministry of Government Services, of inspecting buildings only in response to particular concerns.[8]

The comprehensive schools inspection programmes commenced in the summer of 1979 under the direction of the Ministry of Education.[9] The results of the inspection of schools in Ontario, as of March 10, 1982, were that 80.18% of schools contained no asbestos, 9.35% contained exposed sprayed asbestos, less than 0.4% contained sprayed asbestos in an air plenum, and 10.07% contained other friable asbestos.[10] School boards were asked to take corrective action to eliminate any friable asbestos hazards discovered as a result of the inspections.[11] Options included encapsulation, enclosure, removal, and management and custodial control.[12] Financial assistance to school boards for asbestos control programmes had cost the province approximately $26 million as of February 1983. Further expenditures amounting to $15 million were anticipated at that time.[13]

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[4] U.S., Environmental Protection Agency, "Asbestos; Friable Asbestos-Containing Materials in Schools; Identification and Notification," *Federal Register*, vol. 47, no. 103, 27 May 1982, pp. 23360–23389.

[5] Pinchin, *Asbestos in Buildings*, p. 2.23.

[6] Ibid., chap. 2.

[7] Ibid., p. 2.19.

[8] Ibid., p. 2.23.

[9] Advisory Task Force on Asbestos in Schools, *Report (A Practical Document)* (Toronto: Metropolitan Toronto School Board, May 1980), Appendix A.

The Ontario Ministry of Government Services manages approximately 9,300 buildings in Ontario. Rather than undertake a comprehensive inspection programme, the Ministry of Government Services conducts visual inspections, bulk sampling, and air sampling in response to specific enquiries or concerns. Pinchin reported that as of March 12, 1982, 68 buildings had been inspected for asbestos.[14] Thirty-seven of the 68 buildings were found to contain friable asbestos products, 12 buildings were found to contain no asbestos, and 19 of the buildings were still under investigation. The Ministry of Government Services has responded to the discovery of sprayed asbestos-containing insulation in its buildings with guidelines on management and custodial control. Also, friable pipe insulation is repaired and recovered in accordance with Ministry of Government Services guidelines. These measures have been relatively low cost. It is recognized that any major repair, renovation, or demolition which would disturb friable asbestos-containing material should involve special precautions.[15]

At the federal level of government, Public Works Canada owns and manages almost 13,000 buildings across Canada. These buildings have been reviewed, and 3,700 of them have been physically inspected for sprayed friable asbestos. Only 34 buildings were found to contain sprayed asbestos-containing material. The control options chosen for these buildings were: removal (22 buildings); encapsulation (1 building); enclosure (3 buildings); and management and custodial control (8 buildings). It was estimated that correctional work for these 34 buildings would cost $30.4 million.[16] One can summarize the Ontario experience of Public Works Canada by noting that 23 of its Ontario buildings contained sprayed friable asbestos-containing materials, representing 2.3% of the Public Works Canada buildings in this province. Public Works Canada also leases a number of buildings for various federal government departments; 4.4% of the Ontario buildings leased by Public Works Canada were found to contain sprayed friable asbestos-containing materials.

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[10] Letter from Mr. Stanley Orlowski, Associate Chief Architect, Grants Policy Branch, Ontario Ministry of Education to the Royal Commission on Asbestos, 23 February 1983.

[11] Ontario, Royal Commission on Asbestos, *Proceedings of The Royal Commission on Asbestos, First Public Meeting, Friday, October 31, 1980,* reported by Elizabeth J. Hiscott (Toronto: Royal Commission on Asbestos, 1980), p. 33.

[12] Ibid., p. 32; and G. Bruce Doern, Michael Prince, and Garth McNaughton, *Living with Contradictions: Health and Safety Regulation and Implementation in Ontario,* Royal Commission on Asbestos Study Series, no. 5 (Toronto: Royal Commission on Asbestos, 1982), pp. 4.15–4.16.

[13] Letter from Mr. Stanley Orlowski to the Royal Commission on Asbestos, 23 February 1983.

[14] Pinchin, *Asbestos in Buildings,* p. 2.20.

[15] Ibid., p. 2.21.

[16] Ibid., p. 2.17.

In Chapter 9 we conclude that the potential exposure of building occupants to elevated asbestos fibre levels does not warrant a universal programme of building inspection. Rather, we recommend that if substantial quantities of friable insulation have fallen onto building surfaces and are being disturbed, the building owner should have the material tested to determine whether it contains asbestos.[17] This recommendation is responsive to situations of potential hazard. In the same vein, we recognize that building workers, including custodial, maintenance, renovation, and demolition workers, may be exposed to substantially elevated asbestos fibre levels if their work disturbs friable asbestos in buildings. Accordingly, inspections and testings should occur in the context of work activities which give rise to potential hazards.

We anticipate that inspection in response to potential hazards will allow for timely corrective action, so that potential hazards do not become actual hazards. Such inspection, being selective, can be conducted with due care by qualified experts. In contrast, a crash programme of universal inspection of Ontario buildings could not be conducted at an adequate level of competence because there are few qualified experts in building inspection and because competent analytical laboratories in Ontario do not have the capacity to analyze the bulk samples from a crash programme.

B.2 Visual Inspection

In Chapter 9 we observe that during normal activity, airborne asbestos fibre levels even in "problem" buildings are quite low. However, as Pinchin has pointed out, these readings do not eliminate the possibility of risk in such buildings, since maintenance, renovation, or demolition activities can generate substantial fibre levels for the workers engaged in those activities.[18] As well, damaged asbestos-containing materials can become dislodged and can then be stirred up by cleaning activity or normal building use, generating elevated fibre levels even for building occupants. Accordingly, Pinchin concluded that airborne fibre levels are not an appropriate basis for determining whether to take corrective action in buildings. Rather, the condition of the material and the probability of its being disturbed during maintenance, renovation, or demolition — or by building occupants — are the important factors.[19]

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[17] See Recommendation 9.5.

[18] Pinchin, *Asbestos in Buildings*, pp. 6.15–6.16.

We consider that while air sampling with transmission electron microscope analysis (TEM) can detect elevated fibre levels in a building at the time of air sampling, it does not in general provide a reliable basis for deciding on an appropriate control programme. The main concern raised by friable asbestos-containing material in buildings is the potential elevation of fibre levels during maintenance, renovation, or demolition, which will primarily affect the workers on those jobs. Thus, building inspection should focus on factors which indicate that potential. Building inspection should accordingly consist of a visual inspection and laboratory analysis of bulk samples of friable material thought to contain asbestos.

Factors noted in visual inspections have generally included:

(i) Condition of material: If material is deteriorating, coming loose, or if pieces are falling off, the possibility of fibre release is increased.

(ii) Water damage: If water is leaking onto the material, it can cause rapid deterioration leading to fibre release.

(iii) Exposed surface area: The greater the exposed surface area of the material, the greater the likelihood that any released fibres will reach people working nearby. While a suspended ceiling may cause material to be classified as non-exposed, it should be appreciated that maintenance workers above the suspended ceiling are not protected by the enclosure.

(iv) Accessibility: If the material can be reached by workers or occupants, it is potentially subject to accidental or intentional damage which may release fibres.

(v) Activity and movement: Air movement or building vibration can cause fibre release, while the movement of people or vehicles on the floor can stir up fibres that have settled. Cleaning by dry methods causes particularly high airborne fibre levels.

(vi) Air plenum or direct air stream: If friable material is enclosed within an air plenum, any fibre release which may occur is more likely to be distributed throughout the building than if the material is not enclosed within the ventilating system.

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[19] Ibid., p. 6.16.

(vii) Friability: The more friable the material, the greater the potential for asbestos fibre release. Some wet-applied asbestos-containing materials can be quite hard and release very few fibres under most conditions.

(viii) Asbestos content: The higher the asbestos content in the material, the greater the number of asbestos fibres released under any given condition.

A Task Force of the Council of Safety Associations of Ontario has set out procedures for determining the presence of asbestos hazards. These can be used in investigating the factors listed above.[20]

It is difficult to combine subjective evaluations of the above factors into a single rating of the degree of risk or hazard presented by a particular building. Nonetheless, several attempts have been made to create a single hazard rating, or index, combining all of the factors, for sprayed materials only. These indices have generally been intended to identify potential fibre release so as to permit the ranking of buildings according to this potential. This is achieved by providing a mathematical equation, or algorithm, requiring that each factor be given a numerical rating.

The U.S. EPA published a draft index in 1979 which correlated poorly with measurements of asbestos fibres in the air and which is not likely to be issued in final form.[21] The Ferris Index has been adopted in a modified form by the Toronto Board of Education.[22] The U.S. Navy Risk Evaluation Index considers the number of persons exposed to a particular condition as a factor in assessing a hazard. These indices are discussed by Pinchin.[23] Other than the U.S. EPA index, they do not specify appropriate actions or ranges of actions for any given index level.

Pinchin studied a number of buildings which were inspected and rated according to the various hazard indices and were, at the same time, tested for asbestos content in the air.[24] The purpose of the study was to determine how well the exposure indices could predict or identify buildings in which fibre levels were elevated or, alternatively, how accurately air monitoring could be used to identify buildings which the indices determined were hazardous. In all cases, the asbestos fibre levels were extremely low [less than 11 nanograms per cubic metre (ng/m^3)]. Yet some buildings exhibited conditions which posed the possibility of significant fibre release. Pinchin concluded that there is no useful correlation between the hazard indices and asbestos fibre concentrations measured by him in building air during normal use. This does not, however, rule out the possibility that reference to the hazard indices could identify a building in which fibre levels would be elevated by work activity.

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[20] Task Force of the Council of Safety Associations, *Asbestos: Evaluation and Control of Insulation and Pipe Lagging*, Asbestos in the Workplace — a series of practical guidebooks, no. 8 (Toronto: Council of Safety Associations of Ontario, 1982).

[21] Pinchin, *Asbestos in Buildings*, p. 6.2.

[22] Ibid., p. 1.23. For a discussion of the Ferris Index, see, for example, Karen F. Irving, Rexford G. Alexander, and Harold Bavley, "Asbestos Exposures in Massachusetts Public Schools," *American Industrial Hygiene Association Journal* 41:4 (April 1980): 270–276.

[23] Pinchin, *Asbestos in Buildings*, p. 1.23.

[24] Ibid., pp. 6.6–6.16.

The inspection programme which we will recommend below is responsive to particular types of work activity. (See Recommendation 10.2.) It does not involve reliance on the hazard indices, in the sense that buildings need not be assigned a hazard level rating. Our focus is rather on the detection of friable asbestos before work which might disturb it commences, in order that precautionary steps may be taken. The elements taken into account by the hazard indices, however, remain relevant to the performance of visual inspections.

B.3 Cost of Inspection

The cost of inspecting buildings for asbestos depends on a number of factors discussed by Pinchin.[25] He suggested that a preliminary inspection should be performed to determine whether there is a possibility of asbestos material being present, with a more detailed follow-up inspection to be undertaken only in buildings where this presence seems likely. The preliminary inspection is considerably less costly than a detailed inspection and can greatly reduce overall costs. Pinchin estimated the cost in 1980 Canadian dollars of a preliminary walk-through inspection at about $0.04 per square metre of floor space, while a secondary survey, sufficiently detailed to provide working drawings for contracts for asbestos correction, might cost an average of $0.10 per square metre of floor space.

Table 10.1 shows a summary of inspection costs experienced by Canadian and Ontario government bodies. In most cases the cost per building averaged over all buildings is far less than the cost per building of those buildings that were subjected to a detailed physical inspection. Thus, the Toronto Board of Education and the Rural School Board experienced similar costs in the buildings physically inspected, but the Toronto Board had a much lower cost average over all buildings because a number of buildings did not require a careful secondary inspection. The high cost per square metre of inspecting buildings experienced by the Windsor Utilities Commission is due in part to the small physical size of each building: the cost per square metre of inspecting a building declines as the total floor space in the building increases. In addition, a building owner learns from experience in

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[25] Ibid., chap. 4.

Table 10.1

Summary of Inspection Costs
(1980 $ Canadian)

Organization	Persons Performing Inspection	Cost Per Building ($)		Cost Per Square Metre ($/m²)	
		Averaged Over All Buildings	Averaged Over Buildings Physically Inspected	Averaged Over All Buildings	Averaged Over Buildings Physically Inspected
Public Works Canada	Existing staff	54.20	152.57	0.07	—
National Capital Commission	Existing staff	131.70	131.70	—	—
Toronto Board of Education	Existing staff and Consultant	169.79	536.55	0.02	—
A Rural School Board	Consultant	597.57	597.57	0.10	0.10
Windsor Utilities Commission	Consultant	175.00	175.00	0.34	0.34

SOURCE: Donald J. Pinchin, *Asbestos in Buildings,* Royal Commission on Asbestos Study Series, no. 8 (Toronto: Royal Commission on Asbestos, 1982), Table IV, p. 4.12.

inspecting buildings, so that the cost per square metre declines with the total number of buildings inspected.

B.4 When to Inspect

Friable asbestos-containing materials in buildings may be hazardous to workers who deal with those materials in maintenance, renovation, or demolition work. Such work may also present some hazard to building occupants. Inspection or testing should accordingly be undertaken to determine whether friable asbestos is present prior to doing work on any building in which insulation was installed or replaced prior to 1974.[26] We therefore recommend that:

10.2 The regulation under the Occupational Health and Safety Act on asbestos on construction projects and in buildings should require as follows:

(i) Before the demolition or renovation of any building the characteristics of which raise the possibility that it might contain friable asbestos, the building owner must cause the building or relevant part of the building to be inspected to determine whether friable asbestos-containing materials are present. If friable asbestos-containing materials are present, Recommendation 10.11 should be followed in the case of demolition, and Recommendations 10.14 and 10.17 should be followed in the case of renovation.

(ii) Before performing maintenance or custodial work which would disturb friable fibrous material in any building the characteristics of which raise the possibility that it might contain asbestos, the building owner must cause the material to be disturbed to be tested for asbestos content. If the material is found to contain asbestos, the employer must cause work practices to be adopted which ensure the health and safety of the maintenance or custodial workers, as outlined in Recommendations 10.6, 10.14, and 10.17. Alternatively, the maintenance or custodial work may proceed without the suspect material being tested, provided that the work practices followed would be safe in the event that the material did contain asbestos.

(iii) The requirements of (i) and (ii) above should apply only to buildings in which insulation was installed or replaced before 1974.

A question arises as to who should be responsible for identifying asbestos-containing insulation in buildings and for ensuring that safe work practices are pursued. Under the internal responsibility system of the *Occupational Health and Safety Act,* the worker and employer are jointly responsible for providing safe working conditions. Of equal importance, the building owner is in general responsible for the building and its condition. We believe that the building owner should be jointly responsible with the employer for responding to possible asbestos hazards which may be encountered in the course of work in the building. Accordingly, we have, in the above recommendation, outlined the duties of a building owner to inspect and to test. Similarly, owners should make the results of required inspections and tests known to interested persons. We accordingly recommend that:

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[26] The use of asbestos-containing insulation by and large ceased by 1974. See Chapter 9 of this Report.

10.3 The regulation under the Occupational Health and Safety Act *on asbestos on construction projects and in buildings should require that in any case where a building is required by law to be inspected or tested for asbestos, the building owner must cause the results of the inspection or testing to be recorded as follows:*

(i) in a public place to be designated by the Ministry of Labour;
(ii) in a document maintained on the building premises, under the care of the building owner or his representative. This document should be required to be shown on request to any government inspector, any building occupant, or any worker who is in the building for the purpose of exercising his trade and who might be exposed to friable fibrous material; and
(iii) by placing signs or labels, in areas where asbestos material has been discovered, that clearly indicate the presence of asbestos and warn that the material should not be disturbed without using appropriate precautions.

B.5 Laboratory Procedures for Analysis of Bulk Samples

Two studies conducted for this Commission discussed several cases in the schools programme where confusion arose as to whether or not asbestos was present in a building, specifically as a result of conflicting laboratory reports on bulk materials sent for analysis.[27] This problem demonstrates the importance of the application of careful laboratory procedures for analyzing the material. We therefore recommend that:

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[27] G. Bruce Doern, *The Politics of Risk: The Identification of Toxic and Other Hazardous Substances in Canada,* Royal Commission on Asbestos Study Series, no. 4 (Toronto: Royal Commission on Asbestos, 1982), chap. 2; and Doern, Prince, and McNaughton, *Living with Contradictions: Health and Safety Regulation and Implementation in Ontario,* chap. 4.

10.4 The regulation under the Occupational Health and Safety Act on asbestos on construction projects and in buildings should provide for standardized laboratory procedures to be followed for analysis of bulk material taken from buildings. These procedures should be reviewed from time to time so as to keep up with developing technology.

B.6 Qualifications of Inspectors

Pinchin has noted that ". . . inspections may miss a large percentage of the friable asbestos, unless the inspectors have been adequately trained."[28] In some cases, Pinchin pointed out, improper inspection has led to a failure to detect substantial amounts of asbestos-containing material. Pinchin also documented an instance where inadequate inspection skills almost led to removal of material which was finally determined not to contain asbestos at all.

Pinchin has indicated that given a technical background and some experience in construction, a one- or two-day course would provide sufficient training for building inspection. Such a course would include a description of the uses of asbestos in buildings; a discussion of friability; an introduction to the hazard evaluation index; and techniques for performing inspections, including techniques for taking bulk samples. While the special training required for these inspection functions is not very extensive, it can be readily appreciated that, absent such training, there is a significant risk of missing asbestos in a building. Victims of such errors, moreover, would most often be workers who are not in a position to select or direct the inspector.

Generally, it will be the building owner who hires a building inspector. Given the various reasons which might impel an owner to carry out an inspection, it is not necessarily the case that a building owner would want a thoroughgoing inspection; a more immediate goal might in some cases be to assuage worker or occupant concerns. Alternatively, pursuant to Recommendation 10.2, the objective might be to fulfill a legal obligation to inspect before commencing work on the building. When one considers the expense which can be involved in an asbestos control programme, it becomes apparent that building owners may have a significant interest in not finding asbestos-related risks. Accordingly, the building owner's choice of inspectors should be restricted to suitably qualified persons. Such persons should be under an obligation to carry out inspections competently and honestly.

We note that section 41(1) of the *Occupational Health and Safety Act* provides for the making of such regulations under that Act as are advisable for the health and safety of persons in or about a workplace. Since the quality of inspections for asbestos in buildings would have a direct impact on the health and safety of building workers, it may be possible to pass a regulation pursuant to section 41(1) providing that inspections for asbestos in buildings which are required by law are to be carried out only by suitably qualified persons. In the event that such a provision is believed to be beyond the ambit of section 41(1), an enabling amendment to the *Occupational Health and Safety Act* would be required. We accordingly recommend that:

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[28] Pinchin, *Asbestos in Buildings*, p. 4.2.

10.5 The Ministry of Labour should take steps to institute a register of individuals certified as competent to inspect buildings for asbestos. Certificates of competence should be awarded by the Ministry of Labour upon an applicant's having satisfactorily completed an approved examination. Incompetence or dishonest practices should result in the certificate being revoked. Only individuals named in the register should be permitted to undertake inspections for asbestos in buildings which are required by law.

C. Corrective Actions

There are four primary procedures for controlling asbestos-containing material in a building. These are:

(i) Management and Custodial Control[29] (Deferred Action): Asbestos-containing material is left as is in the building, and procedures are established to reduce or minimize any potential fibre release.

(ii) Enclosure: Asbestos-containing material is enclosed behind a solid barrier.

(iii) Encapsulation: Asbestos-containing material is coated with a bonding agent or sealer.

(iv) Removal: Asbestos-containing material is removed and disposed of.

C.1 Cost of Control Procedures

Pinchin has presented extensive data on the cost of controlling asbestos in buildings, referring primarily to the cost of asbestos removal.[30] He reported that the U.S. EPA determined average costs, in 1980 U.S. dollars, as follows: removal, disposal, and re-insulation — $73.82 per square metre; encapsulation and marking — $24.64 per square metre; and enclosure and marking — $44.23 per square metre. Behind these averages lies a wide range of costs for each of the three control options.

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[29] The term "management and custodial control" is from Pinchin, *Asbestos in Buildings*, p. 1.29.

[30] Ibid., chap. 5.

In Ontario, the cost of removal of sprayed asbestos insulation ranges from a low of $13.72 in 1980 Canadian dollars to a high of $233.78 per square metre, with an average cost range of $50 to $100 per square metre.[31] Prices in 1982 were at the low end of this range.[32] The cost of removing pipe and boiler insulation has been estimated at about $150 per square metre, based on a sample of only two projects. It is not possible to calculate an average cost of encapsulation or enclosure from the Ontario data, but the ratio found in the United States probably applies: 1.0 to 1.8 to 3.0 is the U.S. ratio among the costs of encapsulation, enclosure, and removal.

What would it cost to remove all sprayed asbestos from Ontario buildings? This question is difficult to answer because only a small fraction of all buildings in Ontario have been inspected for asbestos-containing materials, and because there is no single list or inventory of all buildings existing in the province. Based on the inspections that have been performed on schools and government buildings in the province, Pinchin concluded that somewhere between 2 and 10% of the total area of industrial, commercial, institutional, and governmental buildings constructed between 1950 and 1973 in Ontario contain sprayed asbestos material. The most likely percentage is between 4 and 5%. The total floor space of buildings constructed between 1950 and 1973 in Ontario is approximately 45 million square metres. A range of 2 to 10% yields an estimate of 0.9 to 4.5 million square metres of floor space in buildings containing sprayed asbestos-containing materials. The most likely estimate within this range is 1.8 to 2.25 million square metres.[33] In Chapter 9 we estimate the net cost of asbestos removal, including the cost of displacing the present occupants of the building, at about $80 per square metre. Therefore, the total cost of removal of all sprayed material from Ontario buildings would be in the range of $72 million to $360 million with the most likely cost lying in the range of $144 million to $180 million. These figures do not include the cost of removing asbestos-containing pipe and boiler insulation. While no studies were done in this connection, Pinchin has suggested that the cost of removing all asbestos-containing pipe and boiler insulation would approximate the cost of removing all sprayed asbestos material.

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[31] Ibid., p. 5.9.

[32] Personal communication between Dr. Donald J. Pinchin, D.J. Pinchin Consulting Ltd. and Royal Commission on Asbestos Staff, 10 December 1982.

[33] Pinchin, *Asbestos in Buildings*, pp. 3.18–3.19.

It appears, then, that the total cost of removing all friable asbestos-containing material from Ontario buildings might reach half a billion dollars. Such a figure must be taken as a rough approximation because of the great uncertainty about both the amount of asbestos present in buildings and the range of costs for removal.

This estimated cost, however, should be viewed in conjunction with our conclusion that a universal asbestos removal programme is not warranted. Asbestos in buildings presents no hazard to building occupants, except in a limited number of circumstances, specified in Chapter 9, Section E. Cleaning staff and maintenance workers, if our recommendations are followed, can generally use safe procedures to limit their exposure to asbestos fibres. Removal is appropriate only when workers cannot be adequately protected by other means.

C.2 Choosing Among Control Procedures

Let us consider the circumstances in which it would be appropriate to use management and custodial control, enclosure, encapsulation, or removal to deal with an asbestos problem. To do this, each option must be defined and its effects explored.

(a) Management and Custodial Control

The first control option is management and custodial control. Management and custodial control begins with recording the location of any asbestos detected in the building, as outlined in Recommendation 10.2. Regular building workers, including custodial and maintenance staff, should be informed of the possible risks they face, and they should be educated to control these risks. Worker training should include instruction about the known location of asbestos-containing material in the building and about safe work practices. For example, training in wet-cleaning methods, and in the use of special equipment, such as respirators and high efficiency particulate air (HEPA) filtered vacuums, might be appropriate. Equipment reasonably necessary for adequate worker protection should be made available. Locations in the building containing asbestos should be regularly examined; any loose material should be cleaned up, and any damage to the asbestos-containing material should be repaired.

Management and custodial control can best protect workers and building occupants from elevated fibre exposures if appropriate procedures are carefully followed. If the programme is inadequately managed and carried out, it may have no effect whatsoever. Moreover, because the asbestos remains in place, good management must be exercised for the indefinite future, or until the asbestos is removed.

When should management and custodial control be applied? Management and custodial control should always be instituted once friable asbestos-containing material has been identified in a building. It may be a long-term means of preventing significant exposure to airborne fibres, or it may be merely a temporary precaution taken before a removal project is implemented. The scope of any given programme of management and custodial control should depend on the level of hazard created by the asbestos-containing material. For example, if the asbestos is well enclosed or well encapsulated, it is not necessary to train cleaning staff in special cleaning methods. We accordingly recommend that:

10.6 The regulation under the Occupational Health and Safety Act on asbestos on construction projects and in buildings should require that once friable asbestos-containing material is found in a building, the building owner must institute a programme of management and custodial control. The scope of the programme should depend on the level of hazard created by the asbestos-containing material and should provide for the institution of safe work practices, including worker training therein, for all regular building custodial and maintenance workers. The programme should continue until the asbestos-containing material is removed from the building.

To ensure that the scope of any given management and custodial control programme is adequate to protect building workers, we recommend that:

10.7 The regulation under the Occupational Health and Safety Act on asbestos on construction projects and in buildings should require that a written description of any management and custodial control programme, referred to in Recommendation 10.6 above, be submitted to the Ministry of Labour for approval. The Ministry of Labour should be empowered to approve or vary any programme so submitted, in order to ensure adequate protection for building workers.

Non-regular maintenance and custodial work, or maintenance and custodial projects, are dealt with in Section D of this chapter.

While it should be the building owner's responsibility to institute a programme of management and custodial control once friable asbestos is discovered in the building, we recognize that building owners may enter into contracts regarding custodial and maintenance services in their buildings. Accordingly, direct responsibility for worker training and safe work practices under a management and custodial control programme might be shifted from an owner to a contractor. We therefore recommend that:

10.8 The regulation pursuant to the Occupational Health and Safety Act on asbestos on construction projects and in buildings should govern the operations of a contractor who contracts with a building owner to take responsibility for worker training and safe work practices under a management and custodial control programme, so that the contractor would have the same responsibility as the building owner for ensuring that the relevant terms of the management and custodial control programme are met.

It is important that building owners communicate information regarding locations of friable asbestos-containing materials in a building to contractors, so that provisions can be made for worker training and safe work practices. In order to encourage thorough disclosure of potential asbestos hazards by building owners to contractors, it is necessary that every contract for renovation, maintenance, or custodial work specify all known locations of friable asbestos in the building which might be disturbed. Furthermore, in order both to encourage full disclosure by owners and to ensure that it is not contrary to the financial interests of contractors to take precautions for worker protection, the costs of precautions for worker protection against asbestos hazards discovered in the course of the work should be borne by owners. This might be accomplished by way of regulation under the *Occupational Health and Safety Act*, by way of amendment to that Act, or by way of regulation pursuant to an amendment to the regulation-making power under that Act. We accordingly recommend that:

10.9 The Ministry of Labour should take steps to require that every contract for renovation, maintenance, or custodial work specify all known locations of friable asbestos-containing materials in the building which might be disturbed. If, in the course of the work, friable asbestos-containing materials of which the contractor was not informed are discovered in locations which could create some risk to workers, adequate precautions for worker protection should be taken, at the sole expense of the building owner.

Pinchin has suggested that management and custodial control may be a satisfactory long-term approach to friable asbestos-containing pipe and boiler insulation, if no work is to be performed on the material. In our view, this is indeed the most satisfactory approach in most instances because it involves the least disturbance and hence the least exposure. The steps involved would include ensuring that the wrapping surrounding the insulation is intact and painted, to minimize any possibility of fibre release. If the wrapping is damaged, it should be repaired following proper procedures. Management and custodial control may also be a suitable long-term solution for asbestos-containing friable fireproofing insulation which is in good condition, is not damaged, and is not likely to be disturbed.

(b) Enclosure

The second control option is enclosure of the asbestos-containing material. Enclosure can be highly effective in protecting building occupants and workers from asbestos fibre release if the enclosure is carefully constructed and remains intact. If the covering on pipe and boiler insulation is damaged, enclosure which repairs the damaged covering may effectively prevent fibre release, unless work is to be performed on the material. Building spaces which are difficult to access but which contain friable asbestos may be protected by enclosure if it is unlikely that access to such spaces for building work will be required.

Construction of the enclosure itself can generate high fibre levels, requiring worker protection during the installation. (See Recommendation 10.17.) Continued management and custodial control is necessary as the asbestos is still in the building; any breach of the enclosure would raise the possibility of fibre release.

(c) Encapsulation

The third control option is encapsulation. Encapsulation is useful for pipe insulation, which can be wrapped and painted or re-jacketed, and for sprayed insulation, which is in good condition and which can be thoroughly penetrated by the encapsulant. If the sprayed insulation is in poor condition or cannot be thoroughly penetrated by the encapsulant, it may continue to deteriorate after encapsulation, with pieces of encapsulated insulation falling loose. In such cases, the ultimate effectiveness of the encapsulation is greatly reduced, or even negligible. We accordingly recommend that:

10.10 The regulation under the Occupational Health and Safety Act on asbestos on construction projects and in buildings should provide that where removal, enclosure, or encapsulation of sprayed asbestos-containing insulation is contemplated, encapsulation should only be an option if that insulation is in good condition and is sufficiently thin that it can be thoroughly penetrated by the encapsulant, or if removal or enclosure are not practicable.

We note that if sprayed asbestos-containing insulation is sufficiently strong to support an encapsulant and is unlikely to be disturbed by occupants or workers, encapsulation is probably not necessary; the material is unlikely to release significant fibre quantities without encapsulation.

Encapsulation may reduce but not eliminate the problem of friable asbestos in the building. Because the encapsulated material remains in the building, ongoing management and custodial control is required. Then, even when the material is well sealed, building occupants may feel concern, however unwarranted, about the presence of asbestos. Sealed insulation is far more difficult to remove than unsealed insulation. Because the encapsulant does not usually penetrate the entire depth of asbestos-containing pipe insulation, a hazard of fibre release continues to exist for workers who must remove sections of such insulation.

Encapsulation may be useful after asbestos removal procedures to seal down loose fibres that may have been missed. If ceiling tiles are re-used after asbestos removal, they should be cleaned and sometimes coated with a layer of paint or encapsulant to seal in any residual asbestos-containing dust. Encapsulation is also useful in small areas where removal is impractical or re-insulation is not feasible.

(d) Removal

The fourth control option is removal. Removal is highly effective in protecting workers from elevated levels of asbestos fibres, assuming that the removal work is carefully completed. Interestingly, however, the immediate effect of asbestos removal on airborne asbestos fibre levels in buildings is to leave them unchanged or to increase them. Table 10.2 shows the results of air monitoring in buildings containing sprayed friable asbestos insulation material before the start of a removal project, outside the work area during the course of removal, and inside the building after completion of a removal project. Fibre levels inside buildings are generally quite low both before and after removal, but in general are higher immediately after removal than before. Presumably this increase in fibre levels arises from the high dust levels generated during removal and the impossibility of cleaning all dust from a building after a project is completed. One would expect that after some time, whether a period of days or weeks, the fibre levels would decrease. The table also shows that a removal project can cause elevated fibre levels outside the work area if proper controls are not applied.

Pinchin monitored the removal of asbestos insulation from a boiler and some piping, during which great care was taken to minimize airborne fibre concentrations.[34] Water was applied to the insulation being removed, where convenient, in an effort to damp down fibres being released. Even with this wetting, fibre levels were well in excess of 5 fibres per cubic centimetre (f/cc) and may have exceeded 10 f/cc. As work practices were further improved, fibre levels were reduced to a range of 3.8 to 6.1 f/cc. Airborne fibre levels in the vicinity of the project immediately after removal were no lower than before and, in fact, were generally higher. We interpret these data as we do those for sprayed asbestos removal; we assume that within days or weeks after removal, fibre levels would be as low as before. The benefit from removal, then, is not a reduction in average fibre levels, which

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[34] Ibid., pp. 7.21-7.24.

Table 10.2

Air Monitoring Results from Sprayed Asbestos Insulation Projects*

Project Number	Before Start of Project					During Work Outside Area	After Contract Completion				
	Optical Microscopy	Transmission Electron Microscopy				Optical Microscopy	Optical Microscopy	Transmission Electron Microscopy			
	Fibres	Total Asbestos Fibres		Asbestos Fibres >5.0 μm		Fibres	Fibres	Total Asbestos Fibres		Asbestos Fibres >5.0 μm	
	f/cc	f/cc	ng/m^3	f/cc	ng/m^3	f/cc	f/cc	f/cc	ng/m^3	f/cc	ng/m^3
1	0.024	0.040	2.345	<0.006	–		0.06	0.056	0.20	<0.004	–
	0.020	0.010	0.082	<0.006	–		0.04	0.062	2.05	<0.004	–
		0.011	0.044	<0.006	–		0.01	0.052	2.00	<0.005	–
		0.047	0.330	<0.006	–		0.17		0.40	<0.004	–
Average:	0.022	0.027	0.703	<0.006	–	(a)	0.07	0.057	1.16	<0.004	–
2	<0.1	0.024	4.53	0.003	1.4		0.1	0.027	154.2	0.002	120.0
	<0.1	0.017	2.05	0.002	1.6		0.1	0.058	64.2	0.008	53.1
	<0.1	0.019	3.14	0.004	1.8		0.1	0.075	177.3	0.013	148.0
		0.005	1.01	0.002	1.0						
Average:	<0.1	0.016	2.68	0.003	1.5	$0.2^{(b)}$	0.1	0.054	131.9	0.008	107.0
3	<0.1	0.016	2.520	<0.002	–		0.1	0.028	16.5	0.002	14.0
	<0.1	0.011	1.405	0.002	0.1		<0.1	0.036	97.3	0.007	92.0
	<0.1	0.017	1.305	0.002	0.1		<0.1	0.014	60.3	0.007	60.0
Average:	<0.1	0.015	1.743	0.001	0.1	$0.69^{(c)}$	<0.1	0.026	58.0	0.005	56.3
4	<0.1	0.010	1.109	0.004	0.68		<0.1	0.008	2.68	0.002	2.5
	<0.1	0.009	4.309	0.002	2.06		<0.1	0.011	0.40	<0.003	–
	<0.1	0.050	4.600	0.003	1.00			0.010	0.25	<0.003	–
Average:	<0.1	0.023	3.339	0.003	1.25	$2.21^{(d)}$	<0.1	0.010	1.11	0.001	0.8
5	0.001	0.077	(e)	0.004	(e)		0.100	0.14	(e)	0.004	(e)
	0.004	0.020	(e)	<0.001	–		0.007	0.72	(e)	0.080	(e)
	0.004	0.013	(e)	0.002	(e)		0.011	0.74	(e)	<0.001	–
Average:	0.003	0.037		0.002		$0.02^{(f)}$	0.039	0.53		0.028	
6	0.004	0.015	(e)	0.001	(e)		0.028	0.100	(e)	<0.001	–
	0.003	0.014	(e)	<0.001	–		0.019	0.048	(e)	<0.001	–
	0.004	0.007	(e)	0.001	(e)		0.019	0.210	(e)	0.005	(e)
Average:	0.004	0.012		0.001		$0.009^{(g)}$	0.022	0.119		0.002	

Table 10.2 (continued)

Air Monitoring Results from Sprayed Asbestos Insulation Projects*

Project Number	Before Start of Project					During Work Outside Area	After Contract Completion				
	Optical Microscopy	Transmission Electron Microscopy				Optical Microscopy	Optical Microscopy	Transmission Electron Microscopy			
	Fibres	Total Asbestos Fibres		Asbestos Fibres >5.0 μm		Fibres	Fibres	Total Asbestos Fibres		Asbestos Fibres >5.0 μm	
	f/cc	f/cc	ng/m^3	f/cc	ng/m^3	f/cc	f/cc	f/cc	ng/m^3	f/cc	ng/m^3
7	<0.001	0.013	(e)	<0.001	(e)		0.002	0.040	(e)	<0.001	–
	0.003	0.012	(e)	<0.001	(e)		0.003	0.498	(e)	0.034	(e)
	0.002	0.024	(e)	<0.001	(e)		0.004	0.008	(e)	0.003	(e)
Average	0.002	0.016		<0.001		0.008[h]	0.003	0.182		0.012	
8a	0.04	(e)	(e)	0.004	(e)	0.08	0.00	(e)	(e)	0.01	(e)
	0.02					0.3	0.02				
Average	0.03			0.004		0.19	0.01			0.01	
8b	<0.01	0.019	0.13	<0.004		0.04		0.172	1,301.1	0.024	1,200.0
						0.09		0.340	22.9	<0.006	–
Average	<0.01	0.019	0.13	<0.004	–	0.07	0.03[i]	0.256	662.0	0.012	600.0

Notes: * All results from area samples. Measured by NIOSH Method P & CAM 239

(a) No samples taken

(b) Average of 6 measurements ranging from <0.1 to 0.4 f/cc

(c) Average of 9 measurements ranging from <0.1 to 2.8 f/cc

(d) Average of 7 measurements ranging from 0.1 to 8.7 f/cc

(e) Figure not reported

(f) Average of 7 measurements ranging from 0.006 to 0.038 f/cc

(g) Average of 8 measurements ranging from 0.006 to 0.013 f/cc

(h) Average of 12 measurements ranging from 0.002 to 0.16 f/cc

(i) Average of 5 measurements ranging from 0.01 to 0.09 f/cc

SOURCE: Adapted from Donald J. Pinchin, *Asbestos in Buildings*, Royal Commission on Asbestos Study Series, no. 8 (Toronto: Royal Commission on Asbestos, 1982), Table III, p. 7.12.

tend to be low to begin with. Rather, removal prevents elevated fibre exposures for those who must work on or near the asbestos-containing material.

If the asbestos-containing material is in good condition, is not damaged, and is not disturbed by occupants or workers, removal is not warranted. Removal undertaken in these circumstances could increase rather than decrease the total fibre exposure of workers and occupants. In this context it should be noted that wet-applied, or cementitious, sprayed asbestos insulation is more likely to be in good condition than is dry-applied sprayed asbestos insulation. We report in Chapter 9, Section D that Nicholson did not find elevated airborne fibre levels in buildings with wet-applied sprayed asbestos insulation.

C.3 Demolition

We have not found data indicating the fibre levels experienced by demolition workers when dismantling a building or structure that contains friable asbestos. Neither have we seen evidence on asbestos fibre levels in the surrounding ambient air during demolition. We know, however, that fibre levels in the breathing zone of workers removing sprayed asbestos-containing insulation or removing asbestos-containing pipe and boiler insulation were far above the recently adopted Ontario control limits, even with careful work procedures, so that respirators were required for worker protection.[35] In addition, fibre levels outside the work area of an asbestos removal project could be substantially elevated if careful procedures are not followed to seal off the work area. In the absence of other information, we believe it is prudent to assume that demolition may cause as much fibre release as does asbestos removal. Since most buildings with sprayed asbestos insulation and pipe and boiler lagging are now between 10 and 30 years old, the problem of exposure from demolition will be much greater in the future than it is at present.

Two control options are conceivable. One is to remove friable asbestos-containing material before demolition, using safe work practices. The other is to develop procedures that would ensure the health and safety of workers on the demolition site, and of the general public outside, while demolishing a building containing friable asbestos.

We have found no studies that describe procedures adequate for worker protection and the protection of the public when demolishing a building which still contains friable asbestos material. Even if one could saturate the asbestos material with water and enclose the area of the building being demolished, the waste carried out of the building would be a mixture of normal demolition wastes and friable asbestos products. This would all have to be carefully enclosed until safely deposited at a dump site, an extremely difficult task given the huge volume of waste that would be involved. This seems impractical, and we must assume it to be so until studies show otherwise. We, therefore, reject the option of leaving friable asbestos in a building and attempting to control fibre release during and after demolition.

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[35] Ibid., p. 7.25.

U.S. EPA regulations require that friable asbestos-containing material be removed from a building prior to demolition, where more than 80 linear metres of friable asbestos material are to be disturbed.[36] Friable asbestos material is to be wetted prior to removal,[37] and is to be disposed of safely.[38] The EPA is to be notified at least 10 days before demolition of a building containing friable asbestos.[39]

We believe that removal of asbestos just prior to demolition may be considerably less expensive than asbestos removal which is to be followed by reoccupancy. If the building is to be demolished, much less care need be taken to avoid water damage or other damage to furniture and fixtures than if it is to continue to be occupied. Ceiling tiles, lights, and other fixtures that must be removed for asbestos removal need not be replaced afterwards. As well, costs associated with displacing occupants are avoided. We therefore recommend that:

10.11 The regulation under the Occupational Health and Safety Act on asbestos on construction projects and in buildings should require that the owner of a building or structure ensure that prior to the demolition or partial demolition of a building or structure, all friable asbestos-containing materials which might be disturbed are removed from the building or structure, following procedures for removal referred to in Recommendations 10.14 and 10.17.

We note that the removal of asbestos prior to demolition is consistent with the Ministry of Labour's Proposed Regulation Respecting Asbestos on Construction Projects (PRRACP).[40]

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[36] U.S., Environmental Protection Agency, *National Emission Standard for Asbestos*, 40 CFR 61.22(d)(4)(i).

[37] 40 CFR 61.22(d)(4)(ii).

[38] 40 CFR 61.22(j).

[39] 40 CFR 61.22(d)(2).

[40] Ontario, Ministry of Labour, Occupational Health and Safety Division, "Proposed Regulation Respecting Asbestos on Construction Projects and Related Codes," presented at a Public Meeting, 17 January 1983, Toronto. (Mimeographed.) This "Proposed Regulation. . ." is a revision of the "Notice of Proposed Regulation: Designated Substance — Asbestos on Construction Projects," *The Ontario Gazette*, vol. 115-33, Saturday, 14 August 1982, pp. 3194-3197.

Having determined that building demolition must be preceded by removal of friable asbestos, we turn to the problem of determining when asbestos is present. The only reliable way to determine whether friable asbestos-containing material is present in a building about to be demolished is to conduct an inspection of the building prior to demolition, and we have so recommended. (See Recommendation 10.1.) The results of such inspections should be communicated to demolition contractors, so that demolition contracts can provide for the costs of removing asbestos safely, thereby ensuring worker safety. This could be accomplished by way of regulation under the *Occupational Health and Safety Act*, by way of amendment to that Act, or by way of regulation pursuant to an amendment to the regulation-making power under that Act. We accordingly recommend that:

10.12 The Ministry of Labour should take steps to require that every contract for demolition specify all known locations of friable asbestos-containing materials in the building or structure which might be disturbed by the demolition work.

It is always possible that, notwithstanding careful inspection, some friable asbestos-containing material will be discovered during the process of demolition. If the demolition contractor is on a fixed-price contract, it would be financially advantageous to proceed as if the material were not hazardous. The building owner, too, does not have an incentive to deal safely with such material. In order to ensure that it is not contrary to the financial interests of demolition contractors to take such steps as are necessary for the removal of asbestos hazards not specified in the demolition contract, the costs of such removal should be borne by the owners. This could be accomplished by way of regulation under the *Occupational Health and Safety Act*, by way of amendment to that Act, or by way of regulation pursuant to an amendment to the regulation-making power under that Act. We accordingly recommend that:

10.13 The Ministry of Labour should take steps to provide that all contracts for the demolition of a building or structure in Ontario shall be deemed to include a clause which provides for supplemental payment to the demolition contractor, on a time and materials basis, for all reasonable expenses incurred in removing any friable asbestos-containing material which was not specified in the demolition contract.

We anticipate that the construction industry would develop a standard clause embodying the above recommendations, for insertion in all demolition contracts.

D. Safe Procedures for Work with Asbestos

D.1 Recommended Work Practices

Workers and persons in the vicinity of work may be exposed to asbestos as a result of enclosure, encapsulation, or removal of asbestos or as a result of non-control work, such as demolition, renovation, maintenance, or custodial work which disturbs asbestos. The degree of precaution required for any job depends on the dustiness of the job.

There are two ways to approach the protection of building and construction workers. The first approach specifies control limits and requires regular air monitoring to ensure that the control limits are observed. This is the approach, described in Chapter 7, that currently applies in Ontario to all workplace exposures, except those occurring on construction projects. The second approach involves specifying with some precision the procedures which should be followed when performing particular types of work. This is the approach generally used for the occupational health and safety protection of workers on construction projects in Ontario, specified by the Ministry of Labour's Proposed Regulation Respecting Asbestos on Construction Projects and endorsed by us in the introduction to this chapter.

The PRRACP defines a "hazardous work area" as ". . . that part of a project where material containing asbestos is used, handled, dealt with, disturbed or removed and as a result thereof airborne asbestos is likely to be inhaled or ingested by a worker."[41] Two levels of precaution may be required by the PRRACP in a "hazardous work area." In *all* hazardous work areas, the following are required:

(i) The Ministry of Labour must be notified before work begins (section 4).

(ii) Warning signs and barriers must be erected around the work area (section 5).

(iii) Workers must wear respirators and protective clothing (section 6).

(iv) Washing facilities must be made available (section 7).

(v) Eating, drinking, smoking, and chewing materials are prohibited in the work area [section 7(4)].

(vi) The work area must be cleaned regularly and carefully (section 8).

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[41] Ibid., s. 3(1).

(vii) Power tools must have local exhaust [section 9(1)].

(viii) Spraying asbestos is prohibited [section 9(2)].

(ix) Waste material must be carefully packed and disposed of (section 10).

(x) Workers must be properly trained (section 11).

(xi) An asbestos work record must be maintained for each worker (section 14).

(xii) Workers must be subject to medical surveillance (sections 15-18 inclusive).

Where a hazardous work area exists and asbestos is to be stripped, removed, or encapsulated, further precautions are required by Section 13:

(i) The work area must be enclosed and sealed.

(ii) The enclosure must be inspected and repaired daily.

(iii) Asbestos materials to be removed must be wetted, where practicable.

(iv) Exits must have showers and change rooms.

(v) The work area must be carefully cleaned before work commences.

(vi) Before removing the enclosure, the work area must be washed and vacuumed, and all asbestos must be removed or encapsulated.

The definition of "hazardous work area," quoted above, is broad, covering a wide range of activities, including asbestos removal, enclosure, or encapsulation; and renovation or maintenance of buildings or of plumbing, heating, ventilating, air conditioning, electrical or other systems. Any of these activities could be major, disturbing hundreds of square metres of insulation, or small, disturbing only a few handfuls of asbestos. Removing ceiling tiles covering an asbestos-sprayed ceiling, or installing a valve in asbestos-insulated pipe as part of a construction project, might create a "hazardous work area." Furthermore, we have recommended that the PRRACP should be extended to minor maintenance and custodial activities. The variety of "hazardous work areas" will be further enhanced given our recommendation that the Ministry of Labour should frame a regulation that encompasses building maintenance and custodial work as well as construction projects.

Because of the variety of work situations to be covered, we believe that the two levels of precautions envisaged in the PRRACP are insufficient. The general hazardous work area precautions would be excessive for maintenance activities involving little asbestos disturbance and perhaps for encapsulation of pipe and boiler insulation in good condition. It is not plausible that the Ministry of Labour would be notified, barriers erected, workers trained, and worker records maintained every time a hole is drilled in asbestos-cement water pipe, every time an electrician removes a ceiling tile below some sprayed asbestos, or every time a valve on an asbestos-insulated pipe is repaired. Regulatory enforcement, whether on construction projects or in buildings, would be jeopardized if unreasonable burdens are imposed in connection with work which involves minimal asbestos exposure. Accordingly, we propose that three levels of precautions be defined; that the two levels of precautions contained in the PRRACP, with small modifications, become the intermediate and maximum precautions; and that a new set of minimum precautions be devised.

The minimum precautions would require that the worker wear an approved respirator as prescribed in section 1 of the Code for Respiratory Equipment of the PRRACP and that the work area be cleaned using wet methods or a high efficiency particulate air filtered vacuum when work is completed. Eating, drinking, smoking, and chewing materials should be prohibited in the work area, as these interfere with the proper use of respirators. Power tools used on asbestos-containing materials should be equipped with high efficiency particulate air filtered vacuums. Workers should be properly instructed. The Ministry of Labour need not be notified, and exposure records need not be kept. These precautions will be referred to as "Precautions A."

The intermediate precautions should be similar to those described in the PRRACP, in sections 4, 5, 6, 7, 8, 10, and 11 outlined above, with minor modifications. Section 5(b) requires that a hazardous work area be separated from the rest of the project by ". . . the placing of barriers to mark its boundaries." These barriers could be ropes or sawhorses. In accordance with our conclusion that these precautions should not apply to the least hazardous work, we believe that some effort should be made to contain airborne dust within the work area. Accordingly, the hazardous work area should be separated from the rest of the project by a barrier capable of limiting the spread of asbestos fibres outside the work area. An example of such a barrier would be a plastic sheet surrounding the work area, taped to the ceiling, but not sealed at the floor or at entrances.

It is well established that wetting asbestos greatly reduces airborne fibre levels, yet this is not required by sections 4 to 11 inclusive of the PRRACP. In some cases, wetting may be easy and beneficial in reducing dust levels. In other cases, wetting may be impractical because of electrical hazards, damage to furniture or fixtures, or other conditions. We believe that in situations calling for the application of more than minimal precautions, whenever practicable, friable asbestos-containing materials should be wetted before they are disturbed. We will refer to these intermediate precautions as "Precautions B."

The maximum precautions, or "Precautions C," should be as described in section 13 of the PRRACP, outlined above. We are satisfied that these procedures are sufficient both to provide worker protection and to leave the building in a safe condition for further occupancy. They are, however, quite general. Many asbestos control contracts involve specifications which establish precisely how the work is to be performed. An example of such specifications can be found in the Foundation of the Wall and Ceiling Industry's *Guide Specifications for the Abatement of Asbestos Release from Spray- or Trowel-Applied Material in Buildings and Other Structures.* These highly detailed specifications are set out in Appendix H to the Pinchin study.[42] They provide detailed provisions both for protecting workers on the job and for ensuring that the building is in a safe condition when the work is completed. These specifications are consistent with the maximum precautions we have defined. While they are far too detailed for enactment as regulations, they could be used by the building owner to assist in securing competent performance from a contractor. In light of the above, we recommend that:

10.14 The regulation under the Occupational Health and Safety Act on asbestos on construction projects and in buildings should distinguish among three levels of precautions:

"Precautions A":

The minimum precautions, or "Precautions A," should include the following:

(i) Workers must wear respirators in the hazardous work area [section 6, Proposed Regulation Respecting Asbestos on Construction Projects (PRRACP)].

(ii) Power tools used in the hazardous work area on asbestos-containing material may be operated only in conjunction with high efficiency particulate air filtered vacuums (section 9, PRRACP).

(iii) Workers must be educated as to the risks they face when working with asbestos and trained in the appropriate procedures for minimizing those risks (similar to section 11, PRRACP).

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[42] Pinchin, *Asbestos in Buildings*, Appendix H.

"Precautions B":

The intermediate precautions, or "Precautions B," should include, in addition to the precautions set out above as "Precautions A," the following:

(i) *The Ministry of Labour must be notified before the hazardous work begins (section 4, PRRACP).*
(ii) *The work area must be separated from the rest of the project by a barrier capable of limiting the spread of asbestos fibres outside the work area (to be added to PRRACP).*
(iii) *Protective clothing must be worn by all workers (section 7, PRRACP).*
(iv) *Washing facilities must be available to workers (section 7, PRRACP).*
(v) *The hazardous work area must be properly and regularly cleaned (section 8, PRRACP).*
(vi) *No loose asbestos is to remain on the floors (section 10, PRRACP).*
(vii) *Asbestos-containing materials must be wetted prior to disturbing them, where practicable (section 13, PRRACP).*
(viii) *Asbestos work records must be kept for all workers involved in the hazardous work (section 14, PRRACP).*
(ix) *All workers involved in the hazardous work are to be subject to medical surveillance (sections 15–18, PRRACP).*

"Precautions C":

The maximum precautions, or "Precautions C," should include, in addition to the precautions set out above as "Precautions 'A' and 'B,' " the following:

(i) *The work area must be enclosed and sealed (section 13, PRRACP).*
(ii) *Showers and change rooms must be available to all workers (section 13, PRRACP).*
(iii) *The work area must be cleaned prior to the commencement of the work and subjected to a thorough two-stage cleanup after the work is completed (section 13, PRRACP).*

In applying two levels of precautions, the PRRACP distinguishes only between asbestos removal and encapsulation work, on the one hand, and all other construction work where asbestos dust is likely to be inhaled or ingested by a worker, on the other hand. An alternative approach would draw distinctions among types of control work causing high, moderate, and low asbestos exposure risks. For example, encapsulation of pipe and boiler insulation frequently requires only wrapping the existing insulation with fabric and painting the fabric. This may be considerably less dusty than encapsulating sprayed insulation, which requires directing a blast of sprayed paint at the surface. It is far less dusty than asbestos removal. Then, removing a cementitious wet-applied sprayed insulation may be less dusty than removing a dry-applied sprayed insulation. We conclude that different asbestos control projects, including removal, encapsulation, and enclosure, may generate different dust levels and should therefore require different precautions, depending on the type of work.

In addition to the type of work, the size of a particular job may be relevant to the decision as to appropriate work practices. Removal, encapsulation, or enclosure which disturbs only small amounts of asbestos might not require precautions even as strict as the basic PRRACP precautions (our "Precautions B"). We, therefore, propose that control work be classified as small, medium, and large, depending on the area of asbestos to be worked on.

The U.S. EPA recognizes the importance of the size of a job by exempting buildings with less than 15 square metres of sprayed or boiler insulation, or less than 80 linear metres of pipe insulation, from following its work practices regulation.[43] In view of our recommendation that a minimum set of precautions, "Precautions A," be developed, we believe that total exemption is unwarranted. (See Recommendation 10.14.) As well, we propose that the size definition should be in square metres for pipe insulation as well as for sprayed and boiler insulation, since stripping a large diameter pipe will generally generate more dust than stripping a small diameter pipe of the same length. Furthermore, the 15 square metre area of insulation exempted by the U.S. EPA regulations is substantial, and too large in our view to merit classification as "small." We suggest that a small job might be defined as involving 5 square metres or less of insulation surface, and a large job as involving 20 square metres or more. These numbers are, however, a matter of judgement on which the Ministry of Labour should seek advice from industry and labour. We therefore recommend that:

10.15 The Ministry of Labour should consult with industry and labour to develop numerical definitions of areas of insulation to be classified as "small," "medium," and "large" for the purpose of determining which level of precautions, whether "Precautions 'A,' 'B,' or 'C'" referred to in Recommendation 10.14 above, is to be applied to asbestos control work projects of varying types and sizes.

Turning to non-control work, including maintenance, renovation, and custodial work, there may be great variations in asbestos dust levels caused and in the precautions required for worker protection. Several factors might be considered in the determination of the appropriate precautions for a particular project. The extent of contact with the asbestos would be important; direct contact with the asbestos in the location where it was originally applied is generally more hazardous than contact only with asbestos which has fallen from its place of installation, because the quantities involved in the latter case tend to be far smaller than in the former. The larger the area of asbestos to be disturbed, the greater would be the required precautions. If the asbestos is crocidolite or amosite, greater precautions are indicated than if the asbestos is chrysotile, because work with dry crocidolite or amosite is likely to be dustier than work with dry chrysotile and because chrysotile may be easier to wet than the amphiboles.[44] Finally, the higher the asbestos content of the material, the greater would be the required precautions. We therefore recommend that:

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[43] U.S., Environmental Protection Agency, *National Emission Standard for Asbestos*, 40 CFR 61.22(d).

10.16 The Ministry of Labour should consult with industry and labour to develop definitions of major, intermediate, and minor renovation, maintenance, and custodial projects for the purpose of determining which level of precautions, whether "Precautions 'A,' 'B,' or 'C' " referred to in Recommendation 10.14 above, is to be applied in each instance.

Table 10.3 shows how such a regulation might appear. It specifies three levels of precautions, "A," "B," and "C," as defined in Recommendation 10.14. It allows the application of these precautions to differ depending on the type of work and the size of the job or the extent of the hazard. The entries in the table are intended as examples only and not as specific recommendations. Deciding which precautions are appropriate for each type of work and scope of project should require consultation by the Ministry of Labour with representatives of industry and labour. We therefore recommend that:

10.17 The Ministry of Labour should consult with industry and labour to determine, for the purposes of the regulation under the Occupational Health and Safety Act on asbestos on construction projects and in buildings, the appropriate precautions to be applied to large, medium, and small control jobs and to major, intermediate, and minor renovations and maintenance and custodial projects, distinguishing, where appropriate, among wet-sprayed insulation, dry-sprayed insulation, and pipe and boiler insulation.

The three levels of precautions described in Recommendation 10.14 above are designed with a view to *specified* types of work generally per-

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[44] Pinchin, *Asbestos in Buildings*, p. 7.25; and Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Evidence of Mr. Ross Hunt, 16 June 1982, Volume no. 41, p. 77.

Table 10.3

Procedures for Safe Work with Asbestos in Buildings

Procedures (to be applied as indicated below)
A – Minimum precautions
B – Intermediate precautions
C – Maximum precautions

Control Work	**Size of Job**		
	Large	**Medium**	**Small**
Removal			
Pipe and Boiler	C	C	B
Spray – Dry	C	C	B
Wet	C	B	B
Encapsulation, Enclosure			
Pipe and Boiler	B	B	A
Spray – Dry*	C	C	B
Wet*	C	B	A

Renovation, Maintenance, and Custodial Work	**Extent of Work, Dust Level, Fibre Type**		
	Major	**Intermediate**	**Minor**
Renovation			
Pipe and Boiler	Control**	B	A
Spray – Dry	Control	Control	B
Wet	Control	B	A
Maintenance and Custodial Projects	B	B	A
Regular Maintenance	M + CC***	M + CC	M + CC
Regular Custodial Work	M + CC	M + CC	M + CC
Other	A		

Notes: *Encapsulation is often not an appropriate control method for sprayed insulation. See Recommendation 10.10.

**"Control" means removal, encapsulation, or enclosure.

***"M + CC" refers to management and custodial control. See Recommendations 10.6 to 10.9.

formed in buildings and on construction projects. Work situations may occur which cause asbestos dust release and potential hazards to workers and which are not specifically covered by Recommendation 10.17. These work situations should not in general cause very elevated dust levels. Examples of these situations might include work on asbestos-containing drywall joining and taping compounds. To ensure that all potentially hazardous work involving asbestos is subject to regulation, we recommend that:

10.18 The regulation under the Occupational Health and Safety Act on asbestos on construction projects and in buildings should require that types of work in a hazardous work area which are not otherwise dealt with in that regulation shall only be performed in accordance with "Precautions A," described in Recommendation 10.14.

This is indicated in Table 10.3.

"Precautions A" do not include the keeping of asbestos work records or medical surveillance, because "Precautions A" are designed for relatively low asbestos exposure situations. However, if a worker is often exposed to low asbestos exposure situations, the cumulative exposure may be such as to warrant the keeping of asbestos work records and medical surveillance. We accordingly recommend that:

10.19 The regulation under the Occupational Health and Safety Act on asbestos on construction projects and in buildings should provide that where a worker frequently performs work calling for "Precautions A," described in Recommendation 10.14, asbestos work records should be kept for that worker and that worker should be subject to medical surveillance, as if the work called for "Precautions B."

Comments to the Ministry of Labour on the PRRACP questioned the feasibility of enclosing asbestos removal work areas when the material is outdoors, as is the case with exterior piping.[45] The Ministry replied that ". . . if it can be shown that the dispersion of asbestos is being controlled without the use of an enclosure, alternative procedures are allowed under the equivalency section."[46] The equivalency section of the PRRACP is section 19, which states:

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[45] Written submissions to the Ontario Ministry of Labour on the "Proposed Regulation Respecting Asbestos on Construction Projects," by: Canadian Chemical Producers' Association, #969, 26 November 1982; Dow Chemical Canada Inc., #962, 28 October 1982; Asbestos Information Association/North America, #968, 28 October 1982; Du Pont Canada Inc., #954, 29 October 1982; and Ontario Petroleum Association, #971, 6 December 1982.

[46] Ontario, Ministry of Labour, "Asbestos Construction Regulation," text of presentation made in Toronto, Queen's Park, 17 January 1983, p. 29. (Mimeographed.)

> For the purpose of this Regulation, the methods, procedures, composition, design, size or arrangement that may be used may vary from that prescribed in this Regulation or required in the codes issued by the Ministry if the protection or the factors of accuracy and precision afforded thereby are equal to or exceed the protection or the factors of accuracy and precision prescribed in this Regulation or in the codes issued by the Ministry.

An enclosure generally raises the exposure of asbestos workers, lowers the exposure of workers and the general public outside, and prevents the spread of asbestos fibres to the neighbourhood. We understand that currently outdoor asbestos removal is sometimes performed without an enclosure, using large vacuum machines to convey discarded asbestos material and to collect dust, as a local exhaust device. We do not have sufficient information to evaluate this technology. We do, however, believe that working with asbestos outside of buildings will occur with sufficient frequency, particularly in the chemical and petrochemical industries, that the Ministry of Labour should provide more guidance as to what may be acceptable practice. We therefore recommend that:

10.20 The Ministry of Labour should, in consultation with industry and labour, prepare an alternative set of precautions for asbestos removal work outdoors that would allow the substitution of local exhaust ventilation for the use of an enclosure in situations where enclosure is impracticable. This alternative set of precautions should be included in the regulation under the Occupational Health and Safety Act on asbestos on construction projects and in buildings.

We recognize that our recommendations regarding safe work practices on construction projects and on custodial and maintenance projects would at times result in some unnecessary precautions being taken. This is, to some extent, inevitable in any scheme of regulation by procedure designed to apply to very diverse types of work. Nonetheless, flexibility should be maximized, within the bounds of safety. We believe that this could be achieved by providing for Ministry of Labour approval of work procedures which, while varying from those prescribed, are capable of providing adequate worker protection.

The equivalency section of the PRRACP (section 19) permits equivalent protection, involving substitute procedures which are just as protective as those prescribed. While we endorse this provision, we believe that it should also be possible to use substitute procedures that are demonstrated to be *adequately* protective. In discussing control limits for fixed workplaces in Chapter 7, we point out that proper enforcement of such limits means that the average worker will be exposed to one-half or less of the exposure value that a control limit specifies. Outside fixed workplaces, we believe that procedures which produce the same outcome will be adequately protective. Accordingly, when the Ministry of Labour is convinced that substitute procedures, when employed by a trained worker using a reasonable degree of care, will produce the same outcome, it should allow the substitute procedures to be used. We accordingly recommend that:

10.21 The regulation under the Occupational Health and Safety Act on asbestos on construction projects and in buildings should provide that work procedures may be used which vary from those prescribed if the protection afforded by the variant procedures is equal to or exceeds the protection afforded by the prescribed procedures, or if it is demonstrated to the Ministry of Labour that those variant work procedures would always result in average worker exposures which, in the case of chrysotile, are at or lower than one-half the control limit and, in the case of crocidolite or amosite, are at or lower than one-half the exposure values prescribed in Recommendations 7.13 and 7.15. However, the use of variant work procedures should not in any way detract from requirements that would otherwise apply to the particular work situations regarding worker training, Ministry of Labour notification, asbestos waste handling, cleaning of the hazardous work area, wetting of asbestos, asbestos work records, or medical surveillance.

The objective of our recommendations in this section is to maintain worker exposure at levels equal to or less than those allowed by the regulations regarding fixed workplaces, to prevent contamination of the rest of the building, and to leave the building clean after work has been performed. We anticipate that air monitoring would be used from time to time in order to demonstrate the necessity of these precautions for achieving the desired fibre levels. The membrane filter method, using phase contrast microscopy analysis, may be appropriate for assessing dusty work where asbestos fibres are expected to be the primary dust component. For work where much non-asbestos dust is expected, or where fibre levels are expected to be below the limits of reliable phase contrast microscopy analysis, electron microscope methods may be necessary. On some work, both measurement methods should be used simultaneously to add to the understanding of the relationship between them. We accordingly recommend that:

10.22 The Ministry of Labour should conduct air monitoring of control work, renovation work, and of maintenance and custodial work, from time to time, to determine whether the safety precautions referred to in Recommendations 10.14, 10.17, and 10.20 above are sufficient to ensure worker safety or are unnecessarily strict.

The number of workers across Ontario engaged in work which would be covered by a regulation on asbestos on construction projects and on maintenance and custodial projects is enormous, which creates difficulty in setting up a programme directed at identifying the exposed workers and communicating with them directly. Nevertheless, an educated labour force is crucial to the successful implementation of any programme of safe work practices. We recommend that:

10.23 The Ministry of Labour should explore methods of communicating with control, renovation, maintenance, and custodial workers to inform them of the risks they face when working in buildings containing friable asbestos and of the appropriate procedures for minimizing those risks.

D.2 Current Uses of Asbestos-Containing Products in New Construction

The major types of asbestos-containing construction materials in use today from which fibres might be released are asbestos-cement pipe, asbestos-cement sheet, and asbestos insulation board. In his study for this Commission, Professor Gordon M. Bragg reported that asbestos-cement sheet and pipe are infrequently used in Ontario.[47] The major asbestos-cement pipe operation in Ontario closed in 1980, and only a few small plants continue to manufacture asbestos-cement products in the province. It appears, as well, that the use of asbestos insulation board is limited and declining.

Asbestos-cement pipe generally contains both chrysotile and crocidolite asbestos. Crocidolite represented about 20% of the asbestos content of the asbestos-cement pipe manufactured in Ontario by Johns-Manville until its Scarborough pipe operations ceased in 1980.[48] The asbestos content of asbestos-cement sheet is mostly chrysotile, with amosite and/or crocidolite constituting 20% or less of the asbestos content.[49] Asbestos insulation board includes a wide range of products, with both the type and quantity of asbestos varying from product to product. Some asbestos insulation board products consist of almost pure asbestos while others contain only a small percentage of asbestos.[50]

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[47] Gordon M. Bragg, *The Technical Feasibility and Cost of Controlling Workplace Exposure to Asbestos Fibres*, Royal Commission on Asbestos Study Series, no. 7 (Toronto: Royal Commission on Asbestos, 1982), p. 85.

[48] Information submitted to the Royal Commission on Asbestos by Johns-Manville Canada Inc., 1982. See also, William H. Hallenbeck et al., "Is Chrysotile Asbestos Released from Asbestos-Cement Pipe Into Drinking Water?" *Journal American Water Works Association* 70:2 (January 1978): 97.

[49] Personal communication between Dr. Donald J. Pinchin, D.J. Pinchin Technical Consulting Ltd. and Royal Commission on Asbestos Staff, 14 February 1983.

[50] Ibid.

Cutting, drilling, or sawing asbestos-cement materials can release dust which contains asbestos fibres. Table 10.4 shows asbestos fibre exposure levels recorded during work on asbestos-cement sheet and pipe products. Traditional sawing and drilling procedures cause fibre levels well in excess of the new 1 f/cc Ontario workplace control limit and sometimes as much as 20 times this rate. Moreover, to the extent that asbestos-cement products contain crocidolite, the lower crocidolite exposure value envisaged by Recommendation 7.13 would be the proper standard for comparison. Other estimates of fibre levels generated by work on asbestos-cement products may be found in Saheed et al.[51]

Procedures for controlling fibre release during work on asbestos-cement products involve either the use of tools that do not generate significant dust levels or the use of vacuum suction attachments which, when connected to the tools, draw away most dust.[52] However, we have not seen evidence to convince us that the fibre levels which remain are so low as to render the use of respirators unnecessary. The data contained in Table 10.4 suggest that work with asbestos insulation board is dustier than work with asbestos-cement products.

We conclude that "Precautions A," described in Recommendation 10.14 in the previous section, should apply to work with asbestos-cement products and with asbestos insulation board. This is provided for in Recommendation 10.18 dealing with types of work in a hazardous work area not otherwise specifically covered by our recommendations for work procedures on construction projects and on maintenance and custodial projects. We acknowledge that it may be possible to demonstrate that procedures for working with these products which vary from "Precautions A" would, when employed by a trained worker using reasonable care, always result in worker exposures which, in the case of chrysotile, are lower than one-half the control limit and, in the case of crocidolite or amosite, are lower than one-half the exposure values prescribed in Recommendations 7.13 and 7.15. Should this be the case, Recommendation 10.21 would apply.

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[51] W. Saheed et al., "Distribution and Concentration of Asbestos Dust in Commonplace Construction Activities," a project conducted jointly by the University of Toronto, Department of Preventive Medicine and Biostatistics and the Construction Safety Association of Ontario, Research and Development Department, Toronto, 1978, Tables A and B. (Mimeographed.)

[52] A/C Pipe Producers Association, *Recommended Work Practices for A/C Pipe* (Arlington, Virginia: The Association, 1977); American Water Works Association, *Work Practices for Asbestos-Cement Pipe*, Manual 16 (Denver, Colorado: AWWA, 1978); Asbestos Information Association/North America, *Recommended Work Practices for Field Fabrication of Asbestos-Cement Sheet* (Arlington, Virginia: AIA/NA, 1980); and Bragg, *The Technical Feasibility and Cost of Controlling Workplace Exposure to Asbestos Fibres*, p. 86.

Table 10.4

Exposure Levels in Construction: Fibres per Cubic Centimetre

Use of asbestos-cement sheet and pipes	**U.K. Data**	**AIA/NA Data**
Machine drilling	2	1.65
Hand sawing	2-4	0.11
Machine sawing without effective local exhaust ventilation (i) jig saw	2-10	
(ii) circular saw	10-20	20
Machine sawing with effective exhaust ventilation	2	
Compression shearing of pipe		0.2
Use of asbestos insulation board	**U.K. Data**	**Johns-Manville Data**
Drilling vertical structures e.g., column casing	2-5	
Drilling overhead e.g., suspended ceilings	4-10	3.3 – no control 0.1 – with shroud
Sanding and surforming	6-20	
Scribing and breaking	1-5	0.8
Hand sawing	5-12	0.1 – no collection (but dust on floor possible cleanup problem)
Machine sawing without effective local exhaust ventilation (i) jig saw	5-20	2.1
(ii) circular saw	20 upwards	3.0 – with control
Unloading deliveries of board (short-term sampling)		
(i) cut pieces	5-15	
(ii) manufacturers standard size sheets	1-5	

SOURCES: Adapted from: Gordon M. Bragg, *The Technical Feasibility and Cost of Controlling Workplace Exposure to Asbestos Fibres,* Royal Commission on Asbestos Study Series, no. 7 (Toronto: Royal Commission on Asbestos, 1982), Table 3.1, p. 45.

The sources used by Bragg were:

For the U.K. data: U.K., Health and Safety Executive, "Technical Data Note 42: Probable Asbestos Dust Concentrations at Construction Projects," in *Precautions in the Use of Asbestos in the Construction Industry,* a report by the subcommittee of the Joint Advisory Committee on Safety and Health in the Construction Industries (London: Her Majesty's Stationery Office, 1974), p. 8.

For the AIA/NA data: Asbestos Information Association/North America and Association of Asbestos-Cement Pipe Producers, "Recommended Standard for Occupational Asbestos Exposure in Construction and Other Non-Fixed Work Operations," 7 February 1980. (Mimeographed.)

For the Johns-Manville data: Johns-Manville, Research and Development Center, "Evaluation of Tools for the Dust-Free Fabrication of Asbestos Cement," Report no. E411-1050-S1, 9 June 1978; and Report no. E411-1050-S2, 2 August 1978. (Mimeographed.)

If the precautions which we have recommended for asbestos-cement products and for asbestos insulation board are to be effective, it is important that workers know when they are working with these products. It is therefore necessary that these products be labelled, so that purchasers and workers can readily identify them as asbestos-containing materials. We recommend such labelling requirements in Chapter 11, Recommendation 11.1.

Other construction products currently in use that contain asbestos include roof coatings, roofing, caulking, and flooring. The asbestos in the first three product categories is generally saturated with asphalt or tar so that there is virtually no possibility of fibres being released during normal installation and use. The fibres are sufficiently tightly bound that we presume that work with these products would not create a hazardous work area.

In the case of floor tiles, normal installation and use should not cause fibre release. However, Sebastien, Bignon, and Martin have reported an isolated instance in which airborne asbestos fibre levels above background levels were ascribed to severe wear of vinyl-asbestos floor tiles, rather than to damaged friable asbestos-containing materials.[53] The fibre levels observed (8, 21, 25, and 170 ng/m^3) were similar to those in buildings containing sprayed asbestos insulation in good condition. All fibres were less than 3 microns in length and therefore shorter than the fibres we found in Chapter 5 to be most hazardous. The researchers, after a diligent search, could find no asbestos fibre source other than the floor tiles.

The notion that above-background asbestos fibre levels could be caused by vinyl-asbestos floor tile is surprising; the fibres in vinyl-asbestos tiles tend to be tightly bound, and most observers have concluded that the release of respirable fibres from such tiles is most unlikely. However, even if we accept the inference drawn by Sebastien, Bignon, and Martin, the fibre levels generated by severely worn floor tiles were no higher than those found in buildings with sprayed insulation in good condition. We note, performing the conversions suggested in Chapter 9, Section D, that even 170 ng/m^3 would yield only 0.005 f/cc. Accordingly, on the available evidence, we conclude that asbestos floor tiles do not present a hazard to building occupants, or workers, with the sole exception of sanding during removal.

Asbestos floor tiles should not be sanded during removal without "Precautions A," described in Recommendation 10.14, being taken. This should be required by regulation: see Recommendation 10.18, which addresses types of work in a hazardous work area not otherwise specifically covered by our recommendations for work procedures on construction projects and on maintenance and custodial projects. We note that while suppliers of asbestos floor tile usually recommend against sanding, their recommendation may not be generally followed in practice.

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[53] P. Sebastien, J. Bignon, and M. Martin, "Indoor Airborne Asbestos Pollution: From the Ceiling and the Floor," *Science* 216 (25 June 1982): 1412.

Sheet or rolled flooring, as distinct from vinyl-asbestos tiles, often comes with a backing of chrysotile asbestos. Since the backing is under the flooring, no fibres can be released during use. However, when the flooring is removed, some of the asbestos backing usually remains behind on the floor. Suppliers recommend that this backing be removed by wetting and scraping by hand. Sanding is not recommended as it would cause substantial airborne asbestos concentrations. Again, it is suspected that in practice the backing is often removed by sanding. We note that sanding asbestos-containing backing of sheet or rolled flooring would call for the application of "Precautions A" described in Recommendation 10.14: see Recommendation 10.18.

Certain pipe insulation materials containing asbestos are still sold in Ontario. These are discussed in Chapter 9, Section B.2, and include asbestos-insulating cement, corrugated asbestos paper, asbestos tape, and asbestos rope lagging. We are recommending that the application of these products be prohibited: see Recommendation 9.3. In any event, these products are not widely used today.[54]

D.3 Asbestos Control Work Supervisors and Inspectors

It is clear from Pinchin's description of problems encountered in stripping insulation from a pipe and boiler that skill and practice are important ingredients in performing asbestos control work which maintains fibre levels at or below the newly adopted Ontario workplace standard.[55] According to Pinchin, over the course of one removal project, careful attention to work practices reduced fibre levels by a factor of 10.

A number of submissions to this Commission described poorly executed control projects, emphasizing the importance of ensuring that proper work procedures are followed on asbestos control projects.[56] Mr.

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[54] Telephone communication between Mr. Ian Dewar, Dewar Insulations Inc. and Royal Commission on Asbestos Staff, 25 March 1983.

[55] Pinchin, *Asbestos in Buildings*, pp. 7.21–7.25.

[56] RCA Transcript, Submission by Dr. Brian Gibson on behalf of the Toronto Occupational Health Resource Committee, 18 February 1981, Volume no. 3, p. 100; RCA Transcript, Submission by Mr. Bob DeMatteo on behalf of the Ontario Public Service Employees Union, 18 February 1981, Volume no. 3, p. 188; and RCA Transcript, Submission by Mr. Joe Duffy on behalf of the Provincial Building and Construction Trades Council of Ontario, 8 June 1981, Volume no. 7, p. 7.

John Donaldson, speaking on behalf of the International Association of Bridge, Structural and Ornamental Iron Workers, Local 721, in describing a removal project at the Toronto International Airport in 1977, related many examples of work practices which allegedly exposed workers to unnecessarily high asbestos fibre levels.[57] His account of this removal project underscores the inadequacy of spot checks by Ministry of Labour inspectors as the only means of evaluating asbestos-related risks and of verifying that proper control procedures are being carried out.

We note that a draft regulation, "Asbestos Insulation and Coating," was prepared in the United Kingdom in 1981. The proposed regulation would prohibit certain categories of employers and self-employed persons from carrying out work involving asbestos insulation and asbestos coating without a validated licence to do so. The U.K. Health and Safety Commission would be empowered to set the requirements for such licences.[58]

Notwithstanding that procedures for controlling exposure to asbestos may be spelled out in some detail in regulations, informed and adaptive responses to a wide variety of work situations will require specialized knowledge. In order to ensure that this knowledge is available on every asbestos control work site, asbestos control work should be supervised by properly qualified asbestos control work supervisors. This could be provided for by way of regulation under the *Occupational Health and Safety Act,* by way of amendment to that Act, or by way of regulation pursuant to an amendment to the regulation-making power under that Act. We accordingly recommend that:

10.24 The Ministry of Labour should take steps to institute a register of individuals certified as competent to supervise the enclosure, encapsulation, or removal of asbestos-containing sprayed insulation and pipe and boiler insulation. Certificates of competence should be awarded by the Ministry of Labour; incompetence should result in the certificate being revoked.

We further recommend that:

10.25 The Ministry of Labour should take steps to require that no asbestos enclosure, encapsulation, or removal may take place without being supervised by a registered work supervisor. The Ministry of Labour should determine what exceptions to this requirement should be made for small jobs.

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[57] RCA Transcript, Submission by Mr. John Donaldson on behalf of the International Association of Bridge, Structural and Ornamental Iron Workers, Local 721, 18 February 1981, Volume no. 3, pp. 4–19.

[58] In 1982, the Health and Safety Commission decided to adopt these measures, but, as of the summer of 1983, they did not yet have the force of law.

Part V

Asbestos Elsewhere

Chapter 11 Asbestos in the Environment

A. Asbestos in Construction and Consumer Products

A.1 Use and Exposure

Asbestos is commonly found in consumer products. The Asbestos Information Association/North America has suggested that there are over 3,000 different end uses for raw asbestos fibre, many of which find their way into the home.[1] Asbestos may be contained in consumer products as diverse as ironing board covers, oven gloves, toasters, broilers, ovens, refrigerators, and clothes washers and dryers. As well, building materials that are primarily installed by tradesmen, such as asbestos floor tiles and roofing, asbestos-cement sheet, and asbestos-textured paints, may be handled and installed by consumers in a domestic context.

In written submissions made to this Commission, concern was expressed over the potential release and subsequent inhalation of asbestos fibres contained in various consumer products. The Consumers' Association of Canada (CAC) has called for the reduction or elimination of asbestos in consumer and other products, as technology permits.[2]

The degree of hazard posed by an asbestos-containing consumer product is dependent upon the matrix in which the fibre is contained and the frequency and manner of use of the consumer product. This issue was considered by the Consumer Product Safety Commission (CPSC) in the United States. A CPSC memorandum described the responses to a general order issued in December 1980, requiring manufacturers and importers of sixteen categories of products to provide information on the asbestos materials used in their products.[3] The memorandum reviewed the information submitted and raised some concern regarding the release of asbestos fibres contained in only a few products, including clothes washers and dryers, and dishwashers. Although asbestos paper used as a thermal barrier in clothes dryers and dishwashers may emit asbestos fibres as a consequence of mechanical vibration, the CPSC staff concluded that it was unlikely that the asbestos components in these product categories presented a significant health risk. As well, the memorandum referred to evidence that the use of these asbestos components has been discontinued. The CPSC expressed concern ". . . that the asbestos-containing brake lining of some clothes washers is subject to sufficient mechanical stress to release asbestos fibers during use." However, a non-asbestos substitute for these brake linings was actively being sought by manufacturers of these appliances.[4]

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[1] Asbestos Information Association/North America [information pamphlet] (Arlington, Virginia: AIA/NA, 1980).

[2] Consumers' Association of Canada, Written submission to the Royal Commission on Asbestos, #17, January 1981, p. 2.

The findings presented in an earlier report commissioned by the CPSC were consistent with the conclusions drawn from the information submitted in response to the general order.[5] That is, of the 120 different asbestos-containing products evaluated in the earlier report, few seemed to emit asbestos fibres in concentrations which could raise the possibility of any health effect.

Hand-held hairdryers are the only appliances that have been tested in Canada for release of asbestos fibres. Tests undertaken by the Health Protection Branch of Health and Welfare Canada in 1979 revealed that asbestos was released by hand-held hairdryers in concentrations equivalent to normal background levels in the ambient air of Canadian cities.[6] Although the Branch concluded that no health risk was presented by the use of hairdryers containing asbestos liners, the Branch nonetheless recommended that asbestos use in these appliances be discontinued.

The use of asbestos-containing construction products, by householders or by construction workers, including asbestos-cement pipe and sheet, asbestos floor tiles and roofing, and asbestos-textured paints, might involve asbestos fibre release during sawing, cutting, application, or removal. For example, Rohl et al. have reported on the domestic uses of spackling, patching, and taping compounds, many of which contained chrysotile asbestos as a reinforcing agent prior to prohibitions in both Canada and the United States.[7] The fibres become airborne during mixing and sanding of the compounds. The researchers found levels of exposure during home repair work involving the use of such compounds to be as much as 7 to 12 times greater than the 5 fibres per cubic centimetre (f/cc) occupational standard then in force in the United States. Furthermore, the fibres were found to remain suspended after mixing and to move into other rooms.

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[3] U.S., Consumer Product Safety Commission, Memorandum from Rory Sean Fausett, Program Manager, Chemical Hazards Program to the CPSC, Subject, Asbestos General Order, 8 January 1982. (Mimeographed.)

[4] Ibid., pp. 4–5.

[5] A.T. Kearney, Inc., *Review of Asbestos Use in Consumer Products: Final Report*, prepared for the U.S. Consumer Product Safety Commission (Washington, D.C.: U.S. Consumer Product Safety Commission, HIA/Economic Analysis, April 1978).

[6] Canada, Department of National Health and Welfare, Health Protection Branch, Environmental Health Directorate, "Preliminary Study on the Possible Release of Asbestos Fibres During the Operation of Hand-Held Hair Dryers," report prepared by Jean-C. Méranger and Albert B.C. Davey ([Ottawa], May 1979.)

The U.S. National Institute for Occupational Safety and Health (NIOSH), at the request of the CPSC, examined asbestos paper used to insulate hot water pipes, heat ducts, and walls that are in close proximity to stoves and ovens to ascertain the amount of fibre release from these sources. The NIOSH study found that ". . . if folded, torn, or cut, the product released fibers of asbestos to an extent that could produce airborne levels in excess of the NIOSH recommended average level for occupational exposure."[8] This recommended level is 0.1 f/cc, but has not been implemented. It should be noted that the fibre levels released by working with asbestos paper are two orders of magnitude less than the fibre levels Rohl found during the sanding and mixing of drywall compounds.

The major concern with the use of asbestos-containing construction products relates to exposures experienced during application and handling by construction workers. In contrast to construction workers, householders will likely perform such work only infrequently and will generate low cumulative exposure levels. However, considering the relatively intense exposures experienced during improper application of some of these products, and the potential for young children to be exposed to resulting dust, we believe that consumers, as well as construction workers, should be protected by various measures ranging from outright prohibition of certain asbestos-containing products to information about risks and appropriate work practices associated with other products.

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[7] Arthur N. Rohl et al., "Exposure to Asbestos in the Use of Consumer Spackling, Patching, and Taping Compounds," *Science* 189 (15 August 1975): 551-553.

[8] "Commission Authorizes Ban on Asbestos Paper Direct Sales to Consumers and Home Contractors," in *News from U.S. CPSC* (Washington, D.C.: U.S. Consumer Product Safety Commission, 11 April 1980), p. 2.

A.2 Current Regulations

(a) The United States

The *Consumer Product Safety Act*[9] allows the Consumer Product Safety Commission to promulgate consumer product safety standards,[10] to ban consumer products,[11] and to create consumer product safety rules and accompanying product certification and labelling procedures.[12] In December 1977, the Commission issued a ban on consumer patching compounds containing respirable free-form asbestos[13] and a ban on artificial emberizing materials (ash and embers) containing respirable free-form asbestos for consumer use.[14] Under the federal *Hazardous Substances Act,*[15] administered by the CPSC, a ban was placed on general-use garments containing asbestos, except those which have a *bona fide* application for thermal protection and which are constructed so that the fibres will not become airborne.[16]

(b) The United Kingdom

The *Health and Safety at Work Act, 1974,* provides that manufacturers, designers, importers, and suppliers of goods must ensure, so far as is reasonably practicable, that the article is so designed and constructed as to be safe without risks to health when properly used.[17] A voluntary programme of product labelling was introduced in 1976 and applies to all U.K. manufactured asbestos-containing products and waste which could, under foreseeable circumstances, cause a danger to health through release of asbestos. Certain consumer goods were specifically designated for inclusion in the labelling programme.[18]

The European Economic Community agreed to marketing and use directives for asbestos products in June of 1983. These directives incorporate a labelling programme.[19]

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[9] 15 U.S.C.A. § 2051 et seq.

[10] Ibid., § 2056.

[11] Ibid., § 2057.

[12] Ibid., § 2063.

[13] 16 CFR Part 1304, 42 FR 63362, 15 December 1977.

[14] 16 CFR Part 1305.

[15] 15 U.S.C.A. § 2055 et seq.

[16] 15 CFR 1500.17.

[17] 22 & 23 Eliz. II, c. 37, par. 6(1)(a).

[18] Included were oven gloves, oven door replacement seals, ironing board replacement rests, simmering pads, wall plugging compounds, brake linings, insulation board, and cement sheet.

[19] Telephone communication between Mr. Stanley King, U.K. Health and Safety Executive and Royal Commission on Asbestos Staff, 29 June 1983.

(c) Canada — Federal Provisions

The *Hazardous Products Act* prohibits the advertising, selling, or importing of specific hazardous products.[20] Such prohibitions have been imposed on asbestos garments (except those specifically designed for thermal protection and constructed in a manner that ensures that asbestos fibres will not be released);[21] asbestos-containing educational or play products for use by children where they are made in such a way that asbestos may become separated from the products; asbestos material used in modelling and sculpture, drywall joint cements and patching compounds made in such a way that airborne asbestos may become separated from the products during preparation, application, repair, or removal; and products used to simulate ashes and embers.[22] The Canadian regulations appear to cover virtually the same articles as the U.S. regulations.

(d) Canada — The Provinces

While provincial legislative jurisdiction appears to be broad enough to establish hazard standards for asbestos-containing consumer products, the provinces have generally not acted on this jurisdiction to date.[23]

A.3 Recommendations

Although a number of products which presented significant potential for fibre release have been removed from the market by the federal Department of Consumer and Corporate Affairs, we believe that there is a need for a more structured response to the hazards of asbestos-containing construction and consumer products. In fact, the response that has been forthcoming has been erratic. Thus, the voluntary removal of asbestos liners from hairdryers was secured by the federal government in spite of scientific data indicating the absence of any true hazard, while free-form asbestos is currently available at hardware stores throughout the country.[24]

There is little evidence on health risks posed by asbestos-containing products. However, we do know that loose asbestos is patently dusty, while, on the other hand, products in which the asbestos component is thoroughly sealed off or encapsulated are not likely to release asbestos fibres under foreseeable use. Between these two categories of asbestos-containing products are a number of products which could release asbestos dust. Regulation of asbestos-containing products should accordingly distinguish among these three categories of goods. Since both the federal and the provincial governments have legislative power in this area, intergovernmental co-operation is needed to avoid over-regulation and duplication. We therefore recommend that:

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[20] R.S.C. 1970, c. H-3.

[21] S.O.R./73-402.

[22] S.O.R./76-0342, as amended by S.O.R./80-288.

[23] Robert T. Franson et al., *Canadian Law and the Control of Exposure to Hazards*, Science Council of Canada Background Study, no. 39 (Ottawa: Science Council of Canada, October 1977).

[24] Canada, Department of National Health and Welfare, "Preliminary Study on the Possible Release of Asbestos Fibres During the Operation of Hand-Held Hair Dryers."

11.1 The Government of Ontario should take steps, either through federal-provincial collaboration or by having recourse to provincial legislative jurisdiction, towards the regulation of asbestos-containing consumer and construction products in accordance with the following scheme:

Products containing asbestos should be classified into one of three categories:

Category One:

This category would embrace loose asbestos which, during normal use or handling, is likely to release asbestos fibres in significant concentrations. An example of this is loose-fill asbestos insulation.

Category Two:

This category would embrace products containing asbestos which possess the potential to release asbestos fibres in significant concentrations in the event of misuse, improper handling, manipulation such as cutting or sanding, or as a result of product degradation. Products in this category would include asbestos-cement sheet; and asbestos gloves, simmering pads, and ironing board replacement sheets.

Category Three:

This category would embrace products containing asbestos which is sealed off or encapsulated so that the release of asbestos fibres is unlikely under foreseeable use. Products in this category would include most appliances which contain asbestos insulation and molded plastic products containing asbestos filler.

We further recommend that:

11.2 The following regulatory actions should be taken with respect to Categories One and Two:

(i) The manufacture, sale, and use of products in Category One, other than essential products for which there is no adequate

substitute, should be prohibited in Ontario, unless the product is exclusively for the use of a manufacturing enterprise governed by the existing Regulation Respecting Asbestos.

(ii) All products in Category One, the manufacture, sale, or use of which are not prohibited in Ontario, and all products in Category Two, should be labelled with the following warning:
"Caution — Contains Asbestos — Breathing Asbestos May Be Dangerous to Your Health — Consult Pamphlet Available From Vendor For Proper Use."

(iii) All purchasers of construction materials labelled in accordance with (ii) above should be provided with a pamphlet outlining the nature of the hazard posed by the product and the appropriate precautions to be taken in connection with the product.

Finally, we recommend that:

11.3 Products in Category Three should not be subject to regulation.

B. Asbestos in Water, Food, Beverages, and Drugs

B.1 Introduction

In Chapter 5 we discuss the evidence regarding health effects of eating or drinking asbestos fibres. There we conclude that the evidence fails to indicate any increased risk of alimentary tract tumours following the direct ingestion of asbestos fibres. This conclusion is based on two sources of evidence. First, most animal evidence shows that feeding asbestos to animals does not cause an increase in gastrointestinal cancer, and in fact does not cause asbestos fibres to be lodged in the gastrointestinal tract. If the fibres are not retained in the gastrointestinal tract, as they are in the lungs, it is highly unlikely that they will cause disease. Second, epidemiological studies of human health related to asbestos levels in drinking water have generally found no health effects from high asbestos levels. In a Canadian study, Toft et al. analyzed water-borne asbestos levels and mortality rates in 71 municipalities across Canada.[25] The researchers concluded that there was not a significant relationship between water-borne asbestos levels and gastrointestinal cancer. A study by Conforti et al. was the only one of more than a half-dozen studies of health and asbestos in drinking water that suggested any such relationship, and even there the suggested relationship was weak.[26] Only a fraction of the many analyses performed by Conforti et al. pointed to a correlation of asbestos with cancer, and the authors noted that confounding factors such as smoking, occupation, and alcohol consumption may be important but were not allowed for in the study.

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[25] P. Toft et al., "Asbestos and Drinking Water in Canada," *The Science of the Total Environment* 18 (1981): 77–89.

In summary, we find that oral ingestion of asbestos in concentrations currently found in water, food, or beverages in North America is not associated with any significant increase in disease. Although negative epidemiological studies cannot conclusively prove that there is no association, the populations studied have been sufficiently large that all but the smallest health effects would have been detected. For their part, the bulk of the animal studies have shown no association between oral asbestos ingestion and gastrointestinal cancer.

We, therefore, find that there is no reason for public concern about the health effects of asbestos in water, food, and beverages. However, as the presence of asbestos in water, food, and beverages has been extensively studied, we summarize the data below.

B.2 Asbestos in Drinking Water

The measurement of asbestos fibre concentrations in water may be performed using transmission electron microscopy (TEM). The water sample is drawn through a Nuclepore filter, which is then carbon-coated. The filter is dissolved, leaving the thin carbon-coating with embedded fibres ready for examination using a TEM. The U.S. EPA has commissioned a study, to be published by early 1984, which develops a standardized method of measurement for asbestos fibres in water. The fibre concentration is usually reported in millions of fibres of all sizes per litre of water; there is usually no separate count of fibres longer than 5 microns. A comparison of these fibre concentrations with airborne fibre concentrations would be meaningless because serious disease may arise from exposure to airborne fibres, while there is no reason for concern about the health effects of asbestos in water.

Cunningham and Pontefract's Canadian study detected levels of asbestos in tap water, melted snow, and river water ranging from 2 million to 173 million fibres per litre.[27] These results are shown in Table 11.1. Unfiltered tap water in a Quebec asbestos mining town contained the highest levels. River water contained more asbestos fibres than water drawn from a city filtration system and melted snow contained higher amounts than river water. Most fibres detected were below 1 micron in length.

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[26] Paul M. Conforti et al., "Asbestos in Drinking Water and Cancer in the San Francisco Bay Area: 1969–1974 Incidence," *Journal of Chronic Diseases* 34 (1981): 211–224.

[27] Hugh M. Cunningham and Roderic D. Pontefract, "Asbestos Fibres in Beverages and Drinking Water," *Nature (London)* 232 (30 July 1971): 332.

Kay reported on asbestos fibre levels in drinking water from 21 cities in Ontario, drawing on surface waters for samples.[28] Samples were examined at a magnification which ranged from 25,000 to 50,000 times. As with Cunningham and Pontefract's investigation, the detected fibre levels varied widely. For instance, Kay found Ottawa's tap water to have a fibre count of 0.136 million fibres per litre, while Sarnia's count was 3.87 million fibres per litre. Kay's data are summarized in Table 11.2. Additional surveys undertaken in Metropolitan Toronto found levels of asbestos which ranged from 0.724 million to 4.06 million fibres per litre.

Health and Welfare Canada commissioned a national survey for asbestos fibres in Canadian drinking water in 1977.[29] The authors of the study, which was done under the auspices of the Ontario Research Foundation, relied on the U.S. EPA's preliminary interim method to evaluate the concentration and type of asbestos present in water samples. The study reported on samples from 71 locations across Canada, representing the water supplies of close to 55% of the Canadian population. Samples were obtained from the raw water source, from the water treatment plant, and from the water distribution network. The researchers concluded that amphibole asbestos was not a major contaminant of Canadian drinking water supplies. In locations where amphibole asbestos was detected, there was usually a much higher concentration of chrysotile fibres. The highest concentrations of chrysotile fibres were detected in Baie Verte, Newfoundland, and Disraeli, Quebec, at levels of up to 1,800 million fibres per litre. In Ontario, the highest levels were found in Thunder Bay, Kirkland Lake, and Hearst, with detected values of up to 3 million, 3.5 million, and 22 million fibres per litre respectively. Data from this study are shown in Table 11.3. Potable water in the 15 other locations sampled in Ontario had fibre levels below 1 million fibres per litre.

The difficulties in measuring asbestos fibre concentrations in water may be illustrated by the controversy surrounding asbestos levels in the water in Thunder Bay, Ontario, in 1975. Early in 1975, researchers at Lakehead University reported asbestos concentrations in the drinking water in that city ranging from 0.45 million fibres per litre to 14.7 million fibres

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[28] G.H. Kay, "Asbestos in Drinking Water," *Journal American Water Works Association* 66:9 (September 1974): 513–514.

[29] Eric J. Chatfield and M. Jane Dillon, *A National Survey for Asbestos Fibres in Canadian Drinking Water Supplies*, 79-EHD-34 (Ottawa: Health and Welfare Canada, Environmental Health Directorate, 1979).

Table 11.1

Asbestos Fibre Concentrations in Beverages and Water

Sample	Source	Millions of Fibres per Litre
Beer	Canadian 1	4.3
Beer	Canadian 2	6.6
Beer	U.S.A. 1	2.0
Beer	U.S.A. 2	1.1
Sherry	Canadian	4.1
Sherry	Spanish	2.0
Sherry	South African	2.6
Port	Canadian	2.1
Vermouth	French	1.8
Vermouth	Italian	11.7
Soft drink	Ginger ale	12.2
Soft drink	Tonic water I	1.7
Soft drink	Tonic water II	1.7
Soft drink	Orange	2.5
Tap water	Ottawa, Ottawa River*	2.0
Tap water	Toronto, Lake Ontario*	4.4
Tap water	Montreal, St. Lawrence River*	2.4
Tap water	Hull, Quebec, Ottawa River**	9.5
Tap water	Beauport, Quebec, St. Lawrence River (6 km below Quebec City)**	8.1
Tap water	Drummondville, Eastern Townships, Quebec, St. François River*	2.9
Tap water	Asbestos, Eastern Townships, Quebec, Nicolet River*	5.9
Tap water	Thetford Mines, Eastern Townships, Quebec, Lac à la Truite**	172.7
Melted snow	Ottawa, top 30 cm (2-3 weeks' precipitation)	33.5
River water	Ottawa River, at Ottawa	9.5

Notes: *Filtration plant used.
**No filtration plant used.

SOURCE: Hugh M. Cunningham and Roderic D. Pontefract, "Asbestos Fibres in Beverages and Drinking Water," *Nature (London)* 232 (30 July 1971): 332.

Table 11.2

Asbestos Fibre Concentrations in Ontario Tap Water

Sample Location	Source	Millions of Fibres per Litre	Estimated Mass Concentration, Nanograms per Litre
Toronto	Lake Ontario	1.9	0.941
Belleville	Bay of Quinte	0.533	0.937
Brantford	Grand River	0.570	1.13
Brockville*	St. Lawrence River	0.446	0.602
Chatham	Thames River	0.595	1.57
Cornwall	St. Lawrence River	2.11	0.729
Hamilton	Lake Ontario	0.694	0.154
London	Lake Huron	0.456	0.429
Niagara Falls	Niagara River	2.58	2.25
North Bay*	Trout Lake	0.384	0.104
Oshawa	Lake Ontario	0.557	0.159
Ottawa	Ottawa River	0.136	0.093
Pembroke*	Ottawa River	2.85	0.538
Peterborough	Otonabee River	1.86	3.54
Port Colborne	Welland Ship Canal	0.608	0.847
Sarnia*	Lake Huron	3.87	2.13
Sault Ste. Marie*	St. Marys River	0.248	0.141
St. Catharines	Welland Ship Canal	1.03	1.56
Sudbury*	Ramsay Lake	0.297	0.542
St. Thomas	Lake Erie	1.60	0.500
Thunder Bay*	Lake Superior	0.830	0.235
Welland	Welland Ship Canal	0.820	0.479

Note: *No filtration plant used.

SOURCE: Adapted from: G.M. Kay, "Asbestos in Drinking Water," *Journal American Water Works Association* 66:9 (September 1974), Table 1, p. 514.

Table 11.3

Summary of Asbestos Fibre Concentrations in Ontario Tap Water (Millions of Fibres per Litre)

City	Chrysotile			Amphibole			Water Filtration
	Raw Water Input	Treated Water Output	Distribution Network	Raw Water Input	Treated Water Output	Distribution Network	
Cochrane	*	*	0 - 0.5	*	*	0 - 0.5	Yes
Hamilton	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	Yes
Hearst	*	*	11 - 22	*	*	0 - 1.5	No
Kenora	*	*	0 - 1	*	*	0 - 0.5	No
Kingston	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	Yes
Kirkland Lake	*	*	1 - 3.5	*	*	0 - 0.5	No
London	1	0 - 0.5	0 - 1	0 - 0.5	0 - 0.5	0 - 0.5	Yes
Matachewan	0 - 0.5	1	0 - 1	0 - 0.5	0 - 0.5	0 - 0.5	No
Matheson	7.5	1	0 - 1.5	0 - 1	0 - 0.5	0 - 0.5	No
North Bay	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	No
Ottawa	4.5	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	Yes
Peterborough	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	Yes
Sault Ste. Marie	*	*	0 - 0.5	*	*	0 - 0.5	No
Sudbury	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	No
Thunder Bay	2	1	2 - 3	0.5	0 - 0.5	0 - 0.5	No
Tilbury	14**	0 - 0.5	0 - 0.5	0 - 7	0 - 0.5	0 - 0.5	Yes
Toronto	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	0 - 0.5	Yes
Windsor	1.5	0 - 0.5	0 - 0.5	0 - 1.5	0 - 0.5	0 - 0.5	Yes

Notes: *Sample not analyzed.

**High solids content did not permit adequate sensitivity. Result reported corresponds to 2 fibres in 20 grid squares examined.

SOURCE: Adapted from: Eric J. Chatfield and M. Jane Dillon, *A National Survey for Asbestos Fibres in Canadian Drinking Water Supplies*, 79-EHD-34 (Ottawa: Health and Welfare Canada, Environmental Health Directorate, 1979), Table 6, p. 31.

per litre.[30] These data were reported in Thunder Bay and led to demands for investigation and for filtration of the city water supply. Independent tests conducted for the Ministry of the Environment showed fibre concentrations of less than 1 million per litre. At a Thunder Bay City Council meeting in April 1975 it was suggested that the high fibre counts produced by Lakehead University might have been the result of laboratory or analytical errors.

Attempting to resolve the controversy, the Ontario Ministry of the Environment established an inter-laboratory study, sending samples of Thunder Bay drinking water to five Ontario laboratories, including the Ontario Research Foundation, McMaster University, the Canada Centre for Inland Waters, Health and Welfare Canada, and Lakehead University. The last two laboratories did not participate in the study. The first three laboratories did analyze the water, yielding results which were described in a report dated September 1975. The average fibre count reported by the Canada Centre for Inland Waters was 0.63 million fibres per litre; McMaster University, 8.45 million; and the Ontario Research Foundation, 0.06 million fibres per litre.[31] Previous studies had shown the Thunder Bay water to contain less than 1 million fibres per litre.[32] The report suggested that the large inter-laboratory differences in reported fibre concentrations might be attributable to differences in counting techniques and differences in the criteria used to identify asbestos fibres. The Ontario Research Foundation, which used relatively sophisticated means for determining whether fibres were asbestos or some other mineral, reported the lowest fibre count. The report concluded by recommending that standards be set for measuring asbestos fibre concentrations in water. The Ontario Research Foundation has subsequently been engaged by the U.S. Environmental Protection Agency to develop a technique to be used for measuring the asbestos in water in the United States.

Asbestos may be deposited in water supplies by natural mechanisms such as the airborne transfer of fibres from wind erosion of asbestiform mineral outcroppings. Cunningham and Pontefract discussed surveys undertaken in British Columbia and the Yukon which linked the levels of asbestos in water to ground water drainage and surface run-off in areas where there was natural exposure of asbestos-bearing bedrock.[33]

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[30] Don Smith, "University Dean of Science Urges Filtering of Water Now," *Chronicle-Journal* (Thunder Bay, Ontario), 18 April 1975, p. 1.

[31] Ontario, Ministry of the Environment, "An Inter-laboratory Study of Asbestiform Mineral Fibre Levels in the Water Supply of Thunder Bay, Ontario," Rexdale, Ontario, September 1975, Table 7, p. 17.

[32] Ibid., Table 9, p. 22.

[33] Cunningham and Pontefract, "Asbestos Fibres in Beverages and Drinking Water," pp. 332–333.

Another possible source of fibre release into water supplies is the asbestos-cement pipe used for water distribution in sewage systems. Asbestos-cement pipes are composed of 85% Portland cement and 15% asbestos. The asbestos component contains both chrysotile and crocidolite fibres in approximately a 4 to 1 ratio.[34] Sometimes amosite is used in place of crocidolite. The amount of fibre release from interior wall deterioration of asbestos-cement pipe has been the subject of much investigation. Olson addressed this issue and concluded that ". . . water flowing through asbestos-cement pipe does not increase the level of fibre content significantly."[35] In contrast, Buelow, Millette, and McFarren have found that asbestos-cement pipe behaves much like other piping materials, except plastic, that are in common use for the distribution of drinking water. If aggressive conditions towards the piping material exist (measured by pH, alkalinity, and hardness), the pipe will corrode and deteriorate.[36]

Mah and Boatman utilized transmission and scanning electron microscopy to study the interaction between water and asbestos-cement pipe. After one month of water flow, bundles of asbestos fibres were observed on the inner surface of pipe which had originally been smooth. Additionally, aggressive water circulated for 218 days in asbestos-cement pipes exhibited an asbestos content of 3.6 million fibres per litre. Prior to the 218-day period, the asbestos content was 1.35 million fibres per litre.[37]

Substitutes with equivalent performance characteristics are available for asbestos-cement pipe for sewage and water distribution applications. However, for pipe diameters of 24 inches and less, the use of substitutes may not be cost competitive.[38]

It appears that the most important source of asbestos deposition in large cities is industrial. High concentrations of asbestos fibres in water supplies near asbestos mining and manufacturing sites may result from the disposal of industrial asbestos-containing waste. The most notable example arises from the Reserve Mining Company, mining a low grade taconite ore in Babbitt, Minnesota, which is sent to Silver Bay for refining. For every ton of pellets produced, more than 2 tons of silica waste tailings containing cummingtonite are discharged into Lake Superior. Until legal action halted the dumping of the tailings into the lake, the company disposed of 67,000 tons of waste per day. This allegedly caused the concentration of amphibole fibres in Duluth drinking water to rise to between 1 million and 644 million fibres per litre.[39] As well, it was asserted in studies presented during litigation that effluent asbestos particles could move several hundred miles.[40]

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[34] Data from Johns-Manville Canada. See also, Robert A. Clifton, "Asbestos," in *Mineral Facts and Problems*, 1980 ed. (Washington, D.C.: U.S. Department of the Interior, Bureau of Mines, 1981), pp. 1–17.

[35] Harold L. Olson, "Asbestos in Potable-Water Supplies," *Journal American Water Works Association* 66:9 (September 1974): 515–518.

[36] R.W. Buelow, J.R. Millette, and E.F. McFarren, "Field Investigation of the Performance of Asbestos-Cement Pipe Under Various Water Quality Conditions," Cincinnati, Ohio, U.S. Environmental Protection Agency, 1977.

[37] M. Mah and E.S. Boatman, "Scanning and Transmission Electron Microscopy of New and Used Asbestos-Cement Pipe Utilized in the Distribution of Water Supplies," in *Scanning Electron Microscopy*/1978/I, ed. O. Johari (AMF O'Hare, Illinois: SEM Inc., 1978), pp. 85–92.

[38] Richard A. Simonds and James L. Warden, "Substitutes for Asbestos-Cement Pipe," in *Proceedings of the National Workshop on Substitutes for Asbestos*, Arlington, Virginia: 14–16 July 1980, EPA-560/3-80-001 (Washington, D.C.: U.S. Environmental Protection Agency, 1980), p. 160.

We are not aware of asbestos wastes in Ontario being discharged into fresh water lakes in quantities approaching those discharged by Reserve Mining. In any event, we have concluded that the evidence fails to indicate adverse health effects from asbestos fibres in water.

In the United States, the Asbestos Manufacturing Point Source Category Regulations,[41] promulgated under the authority of the *Federal Water Pollution Control Amendment Act* of 1972,[42] limit pollution discharge, including total suspended solids, pH, and chemical oxygen demand, for effluents from various asbestos sources. The Act requires that all industrial sources treat effluents by applying the *best practicable control technology* (BPT) available by July 1, 1977 and the *best available control technology economically achievable* (BAT) by July 1, 1984. The BPT and the BAT are both defined for various asbestos manufacturing concerns. It is not anticipated that these targets will be achieved.

While the U.S. Food and Drug Administration has statutory authority to protect the public from unsafe hazards, no regulations governing levels of asbestos in water have been passed.

In the United Kingdom, the *Water Act, 1973*, requires local authorities to supply "wholesome" water.[43] The Model Water Byelaws, 1966, promulgated under this Act, prohibit allowing materials which can cause contamination to come into contact with water.

In Canada, the federal government has not established standards regulating asbestos-containing effluents. Most provinces have enacted water quality legislation. In most cases, these statutes contain general prohibitions preventing the deposit of substances in water which degrade water quality.

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[39] R.W. Durham and Thomas W.S. Pang, *Asbestos Fibers in Lake Superior*, American Society for Testing and Materials: Special Technical Publications, no. 573 (Philadelphia: ASTM, 1975).

[40] *U.S. v. Reserve Mining*, 380 F. Supp. 11; 6 ERC 1657 at 1669 (1974).

[41] 39 FR 7526, 26 February 1974.

[42] 33 U.S.C.A. § 1251.

[43] 21 Eliz. II, c. 37, s. 11(2).

Effluent discharge into water is, in Ontario, subject to the provisions of the *Ontario Water Resources Act*, which prohibits the deposit of any material which may cause injury to any person, animal, bird, or any living thing.[44] However, regulations promulgated under the Act do not set specific effluent standards for asbestos. The "Ontario Drinking Water Objectives," developed by the Ministry of the Environment, state that it is not possible at present to establish a standard for asbestos levels in drinking water, in view of the lack of epidemiological data.[45]

In view of our conclusions set out at the beginning of this section that the evidence fails to indicate adverse health effects from oral ingestion of asbestos, we do not recommend any change in the Ministry of the Environment's present approach to asbestos in drinking water. The health evidence does not suggest a need for standards for asbestos levels in water at this time.

B.3 Food and Beverages

Asbestos has been widely used as a component of filters employed by the food industry. Cunningham and Pontefract measured the amount of asbestos in filtrate using electron microscope methods and found these levels to be comparable to those in tap water, melted snow, and river water.[46] For the Canadian samples, all the asbestos identified was chrysotile, with a length less than 1 micron. The study found between 1.1 million and 6.6 million asbestos fibres per litre in Canadian and American beer and between 1.7 million and 12.2 million fibres per litre in Canadian soft drinks.

Wehman and Plantholt detected asbestos in commercial gin.[47] Gaudichet et al. studied asbestos fibres in 42 bottles of wine from France and abroad and found statistically significant concentrations of chrysotile asbestos in 15 bottles. Concentrations ranged from 2 million to 60 million fibres per litre with a fibre length of from 0.9 to 3.9 microns.[48]

In June 1977, the Consumers' Association of Canada (CAC) published findings similar to those cited above. According to tests conducted by the CAC, levels of asbestos in excess of 2 million fibres per litre could be detected in foreign wines. The CAC asserted that "The presence of any asbestos in wine is unnecessary and dangerous" and recommended ". . . prohibition of the use of asbestos filters in preparation of any material which would find its way into the human body. . . ."[49]

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[44] R.S.O. 1980, c. 361, ss. 14, 15(3), 16(1), and 16(3).

[45] Ontario, Ministry of the Environment, Water Resources Branch, "Ontario Drinking Water Objectives," Toronto, in press, 1983.

[46] Cunningham and Pontefract, "Asbestos Fibres in Beverages and Drinking Water," p. 332.

[47] Henry J. Wehman and Barbara A. Plantholt, "Asbestos Fibrils in Beverages. I. Gin," *Bulletin of Environmental Contamination and Toxicology* 11:3 (March 1974): 267–272.

[48] A. Gaudichet et al., "Asbestos Fibers in Wines: Relation to Filtration Process," *Journal of Toxicology and Environmental Health* 4:5-6 (September-November 1978): 853–860.

However, the CAC study and the other beverage surveys cited above did not show that asbestos filters were responsible for the asbestos contamination in beverages and water. In order to identify the filter as a source of fibre emission, it would have been necessary to demonstrate that the asbestos levels found in water used for beverage production were significantly lower than the levels detected in the final product. However, none of the studies above presented such measurements. The Health Protection Branch of Health and Welfare Canada does not consider action restricting the use of asbestos as a filter component to be necessary on the basis that ". . . it does not appear that the use of asbestos component filters results in levels of asbestos fibres in the finished product above natural background levels."[50] We agree with this conclusion.

We note, however, that in Ontario, the Liquor Control Board (LCBO) reacted to the publication of the CAC report by issuing a directive calling for the immediate cessation of the use of asbestos filters by domestic and foreign producers of wines, spirits, and beer.[51]

In its submission to this Commission, A.O. Wilson Process Equipment Limited, a filter manufacturer, charged that the LCBO has enforced the directive in a fashion which imposes severe restrictions on Ontario wine producers while turning a blind eye to violations committed overseas:

> Wine filtration in each and every major wine producing country of the world is, to our knowledge, using the finest filtration material available — "asbestos," and their respective products are being imported into the province of Ontario and sold through our LCBO to the public. But it's a *no no* for Ontario wineries to use this identical material to filter their wines.[52]

Our staff has determined that only a few samples are examined by the LCBO each year, out of millions of bottles sold and hundreds of brands listed. Although foreign manufacturers are informed of the directive, it is reasonable to assume that without greater diligence in monitoring and enforcement, these overseas manufacturers will enjoy a wide degree of latitude in complying. It therefore appears that the use of asbestos filters by foreign producers continues unimpeded.

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[49] "Test: Asbestos in Wine," *Canadian Consumer* (June 1977): 44–47.

[50] Sandra Glasbeek, *A Survey of Asbestos Policies in Canada with Particular Emphasis on Ontario*, Royal Commission on Asbestos Background Paper Series, no. 1 (Toronto: Royal Commission on Asbestos, 1981), p. 40.

[51] Ibid.

[52] A.O. Wilson Process Equipment Limited, Written submission to the Royal Commission on Asbestos, #58, 1981, p. 2.

Regarding the adequacy of substitute materials, it appears that the filters containing non-asbestos substitutes, such as cellulose and glass, are equal in performance to asbestos filters, save for the removal of "haze" from liquid beverages, an important limitation. These non-asbestos filters, which can be used interchangeably with asbestos filters, are reported to cost 10 to 15% more than asbestos filters.[53]

Other jurisdictions have not imposed comprehensive regulations on asbestos in food and beverages. In the United States, consideration was given to regulating the use of asbestos in talc used as a food or ingestable drug ingredient, but action was deferred until further evidence on the effect of asbestos ingestion was available.

In the United Kingdom, the *Food and Drugs Act, 1955*, provides that no substance may be added to food that would render it "injurious to health."[54] However, with one minor exception, no regulations have been enacted which specifically address the question of asbestos in food. The one exception is in the Miscellaneous Additives in Food Regulation, 1980, which provides that asbestos should not be present in food talc.[55] The U.K. Advisory Committee on Asbestos rejected specific statutory control of asbestos in food, but recommended a review of information concerning the risk to health from the contamination of food and drink by asbestos. The Advisory Committee also reported the recommendation of a Food Additives and Contaminants Committee that attempts should be made to find alternative materials for asbestos filters used in the preparation of food.[56] Apparently, the great majority, if not all, uses of asbestos filters in the preparation of food and drink have now been phased out in the United Kingdom. This was accomplished by industry, with the encouragement of government.[57]

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[53] GCA Corporation, "Asbestos Substitute Performance Analysis," draft revised final report prepared by Nancy Krusell and David Cogley for the U.S. Environmental Protection Agency, GCA-TR-81-32-G (Bedford, Mass.: GCA Corporation, February 1982), pp. 52–63.

[54] 4 Eliz. II, c. 16, s. 1(1).

[55] S.I. 1980/1834.

[56] U.K., Advisory Committee on Asbestos, *Asbestos — Volume I: Final Report of the Advisory Committee* (Simpson Report), William J. Simpson, Chairman (London: Her Majesty's Stationery Office, 1979), paragraphs 260–263 and Recommendation 39, pp. 92–93; and paragraphs P23–P24, p. 95.

[57] Telephone communication between Mr. Stanley King and Royal Commission on Asbestos Staff, 29 June 1983.

At the federal level in Canada, the *Food and Drug Act* provides that no person shall sell an article of food that has in it a "poisonous or harmful" substance.[58] No regulations have been passed under this Act which would restrict the use of asbestos in the food industry.[59]

In most provinces, provincial public health legislation contains provisions for control over food. Food unfit for human consumption may be prohibited under such legislation. The Ontario *Public Health Act* provides that local authorities may regulate the maintenance of premises where food or beverages are being produced.[60] The Food Premises Regulation under the *Public Health Act* provides that premises where food is handled must be free from any condition that may be "dangerous to health."[61] However, the only Ontario agency to prohibit the use of asbestos in food and beverage preparation is the Liquor Control Board, which, as discussed above, has prohibited the use of asbestos filters by producers of wines, spirits, and beer.

In view of the evidence summarized above on the health effects of ingested asbestos, and in view of the fact that asbestos fibre levels in filtrate do not appear to be significantly higher than levels in drinking water, we see no need for new legislation which would specifically limit levels of asbestos in food and beverages. In the same vein, because there is no evidence that asbestos filtration of wines, spirits, and beer causes health problems or that asbestos filters raise the asbestos concentration in beverages, and because the LCBO ban on asbestos filters is not and cannot be enforced effectively against foreign producers, we recommend that:

11.4 The Ministry of Consumer and Commercial Relations should take steps to repeal the Liquor Control Board of Ontario ban on the use of asbestos filters.

B.4 Drugs

Drugs which are injected directly into the body should be considered as posing potentially different concerns than do materials which are inhaled or ingested. Nicholson, Maggiore, and Selikoff examined samples of parenteral (i.e., injectable) drugs in order to determine if they contained asbestos fibre concentrations greater than those in distilled water used in reconstitution. One-third of the samples from two sets of 17 widely used parenteral drugs were found to have levels of chrysotile in excess of those found in distilled water. The researchers linked the chrysotile contamination to the use of chrysotile-containing filters in the manufacture of drugs.[62]

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[58] R.S.C. 1970, c. F-27, s. 4(a).

[59] Food and Drug Regulations, CRC, Vol. VIII, c. 870, p. 5963.

[60] R.S.O. 1980, c. 409, s. 9, pars. 20, 21, 38, 39.

[61] R.R.O. 1980, Reg. 840, s. 12(a)(i).

Selikoff and Lee have reported on a follow-up study undertaken in 1974 to determine whether contamination was a continuing problem with injectable drugs and whether it occurred in oral drugs. Of the 49 parenteral drugs sampled, 8 were found to have concentrations of asbestos at least 10 times greater than the average background levels in terms of both number and mass of fibres present. None of the oral drugs showed significantly high concentrations of asbestos.[63]

In the United States, a study was undertaken on the carcinogenic effects of intravenous injection of small fibres of chrysotile asbestos into rats and mice. The Research Project Summary concluded as follows:

> The studies have demonstrated that by the intravenous route the administration of fairly large doses of chrysotile asbestos to standard strains of mice and rats on an acute and subacute basis can be tolerated, and have little effect on survival rate. At large doses, up to about 1.6 x 10^{10} fibers/kg, over a period of 4 weeks, no carcinogenic effects were demonstrated in rats when studied for a lifetime. On the other hand, whereas mice survived well, there was evidence of carcinogenicity that was dose related and time related and possibly sex related. Whereas there were not enough animals on test to demonstrate a "no effect" dose, there is a suggestion that this dose would be fairly high, perhaps as high as 8 x 10^8 fibers/kg. Of course there is no way of extrapolating such figures from mouse to man, and man frequently has a body burden (lung) from the inhalation route. It would seem prudent to avoid exposure to chrysotile asbestos in parenteral products whenever possible, and this has been done in the FR Final Order dated March 14, 1975.[64]

Comparing the huge doses administered in the U.S. study to the trace asbestos found in filtered drugs leads us to conclude that the risk of cancer caused by the injection of drugs is negligible.

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[62] William J. Nicholson, Carl J. Maggiore, and Irving J. Selikoff, "Asbestos Contamination of Parenteral Drugs," *Science* 177:44 (14 July 1972): 171–173.

[63] Irving J. Selikoff and Douglas H.K. Lee, *Asbestos and Disease* (New York: Academic Press, 1978), pp. 128–130.

[64] U.S., Food and Drug Administration, National Center for Drugs and Biologics, Research Project Summary of FDA 223-77-3017, and prior contracts entitled "Animal Studies of Chrysotile Asbestos by the I.V. Route," prepared by International Research and Development Corporation, Mattawan, Michigan, 18 December 1980, p. 119. See also, 40 FR 11865-11869, 14 March 1975.

The U.S. Final Order of March 14, 1975, referred to above, prohibits the use of asbestos filters in the manufacture, processing, or packaging of parenteral drugs, unless it is not possible to manufacture that drug without the use of such a filter.[65] If use of an asbestos filter is necessary, an additional non-fibre releasing filter must be used unless it is proved that such additional filtration would compromise the safety or effectiveness of the drug.

In Canada, drugs are regulated by the federal government under the *Food and Drug Act*.[66] There is no regulation regarding the presence of asbestos in drugs or the use of asbestos in the manufacture of drugs.

The provinces may regulate asbestos contamination in drugs by virtue of their capacity under the Constitution to protect public health. In some provinces, specific provision is made for regulation of the quality of drugs, either through public health legislation or through legislation governing pharmaceuticals. In Ontario, the *Public Health Act* allows the Ministry of Health to control the sale of impure vaccines and serums.[67] We are not aware of any provincial law directed specifically at asbestos in drugs.

The Health Protection Branch of Health and Welfare Canada has informed the Commission that it is aware of only two applications in which asbestos may be present in the manufacture of drugs in Canada. The Salk vaccine, administered subcutaneously, is filtered with an asbestos filter and is subsequently filtered two more times, first with a nylon filter and then with a Millipore filter. The Sabin vaccine, given orally, is filtered with an asbestos filter and then with a nylon filter. The secondary non-asbestos filters serve to reduce the asbestos content in the drugs. Manufacturers of these drugs are currently attempting safely to eliminate the use of asbestos filters; this may be a few years away.[68]

We see no need for regulatory action to reduce the existing use of asbestos filters in parenteral drugs in Canada.

C. Asbestos in the Ambient Air

C.1 Measurement

Measuring asbestos fibre concentrations in the ambient air, like measuring such concentrations in buildings, is difficult because the asbestos fibre concentrations are very low and because air sampling filters may collect a substantial amount of non-asbestos particles and fibres which must be differentiated from asbestos. In Chapter 9, Section C we discuss methods for measuring asbestos in the ambient air and in buildings and problems associated with those methods, and we reach the following conclusions. Optical microscope analysis of membrane filters, widely used for measuring fibre levels in the workplace, is not useful for measuring low concentrations of asbestos fibres, since ambient concentrations are often below the detection limit of 0.1 f/cc and because the optical microscope cannot distinguish between asbestos and non-asbestos fibres. Accurate measurement requires the use of an electron microscope, preferably a transmission electron microscope (TEM).

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[65] 21 CFR Part 133.

[66] R.S.C. 1970, c. F-27.

[67] R.S.O. 1980, c. 409, s. 7(c).

[68] Telephone communication between Dr. John Furesz, Director, Bureau of Biologic Drugs, Health Protection Branch, Health and Welfare Canada and Royal Commission on Asbestos Staff, 16 May 1983.

The TEM is used to count and identify fibres in a sample, after which the results may be reported either in terms of fibres per cubic centimetre (f/cc) or in terms of the mass of asbestos, measured in nanograms per cubic metre (ng/m^3), or both. If only the mass data are reported, the process of attempting to estimate from those data the number of fibres per cubic centimetre is fraught with problems. (See Chapter 9, Section D.) Nonetheless, such attempts provide the only means of comparing TEM mass data to the data available on asbestos fibre levels in the workplace, since these latter data are generally presented in terms of fibres per cubic centimetre.

Because the TEM can resolve smaller objects than can the optical microscope, the TEM will make it possible to detect more fibres in a given air sample than would an optical microscope applied to the same sample. As a rough approximation, the TEM would detect 10 times as many fibres longer than 5 microns as would an optical microscope. Thus, TEM counts of fibres longer than 5 microns may be divided by 10 to determine approximately how many fibres might have been counted by the optical microscope, which will be referred to hereafter as the "optical microscope equivalent" of the TEM fibre count.

Finally, a single standardized method of measuring and analyzing ambient air samples has not yet been developed. Thus, it is difficult to compare measurements taken by different researchers and difficult to interpret the results that are available.

It is interesting to review the Ontario ambient asbestos guideline in the light of these measurement problems. The Ontario Ministry of the Environment's Air Resources Branch has adopted a guideline which sets out a 24-hour ambient air objective of 0.04 f/cc over 5 microns in length. The Ministry usually tests for compliance using the TEM, although the Ministry would consider using another sort of microscope if it were shown to be equally accurate.[69]

At the time this ambient air guideline was developed, the occupational exposure standard was 2 f/cc longer than 5 microns measured by the optical microscope. The work week is approximately 40 hours, while exposure to ambient air could be continuous, or 168 hours per week. Thus, to maintain the same cumulative exposure, the environmental standard would have to be less than one-fourth the workplace standard, 0.476 f/cc measured optically. Because environmental exposure is involuntary and uninformed, and may affect the infirm as well as the healthy, a lower exposure was deemed appropriate, so the exposure was divided by 10. Rounding this downward yielded the 0.04 f/cc ambient standard. However, compliance with the environmental guideline requires use of the TEM, so a measurement of 0.04 f/cc by TEM might be equivalent to approximately 0.004 f/cc measured by the optical microscope. It does not appear that this greater resolution of the TEM was considered in setting the Ontario environmental guideline, with the result that the guideline may provide an order of magnitude more protection than was contemplated. Exposures that comply with the environmental guidelines may thus be approximately 1/250 of the chrysotile exposure allowed by the recently adopted workplace control limit. However, as noted above, no single measurement methodology has been promulgated for applying this guideline. The Ministry itself uses TEM, and this of course is appropriate, while the use of optical microscope methods is completely inappropriate.

C.2 Airborne Asbestos Concentrations

Asbestos fibre concentrations in the air have been studied in locations outside Ontario. Chapter 9, Table 9.4 summarizes air monitoring data in buildings and outdoors. Sebastien's study of air quality in outdoor locations in Paris, using electron microscope techniques, found average mass concentrations of 0.96 ng/m^3, with 99% of all observations at or less than 7 ng/m^3. Sebastien did not report the fibre count. Nicholson studied ambient asbestos fibre concentrations in the outdoor air in New York City, using the TEM and a preparation technique called the "rub-out method." Eighty-three percent of Nicholson's observations were at or below 20 ng/m^3, and 96% were less than 50 ng/m^3. In Chapter 9 we estimate the optical fibre count that might be implied by these mass measurements and thereby equate Sebastien's 7 ng/m^3 to 0.0002 f/cc measured optically, while Nicholson's 20 ng/m^3 is equivalent to 0.0006 f/cc measured optically. We conclude that in the outdoor air it would be quite unusual to observe more than the optical microscope equivalent of 0.001 f/cc longer than 5 microns.

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[69] Telephone communication between Mr. Bruce Martin, Air Resources Branch, Ontario Ministry of the Environment and Royal Commission on Asbestos Staff, 8 July 1983.

Selikoff, Nicholson, and Langer found concentrations of 10 to 50 ng/m^3 of chrysotile in the air of New York City in an early study published in 1972.[70] Lanting and den Boeft used the indirect preparation method and TEM analysis to determine asbestos fibre concentrations in industrial and rural towns in Holland.[71] They found, in large industrial towns, levels of 0.5 to 2.0 ng/m^3 and in rural towns, levels of 0.1 to 0.5 ng/m^3. The fibre concentrations were higher in a tunnel carrying heavy traffic, a result the authors attributed to dust from brake linings. Bruckman and Rubino reported that ambient chrysotile asbestos levels were generally less than 10 ng/m^3 in both urban and rural locations in Connecticut.[72] However, measurements conducted near highway toll plazas indicated that levels higher than 10 ng/m^3 occurred, and it was concluded that brake lining wear was responsible.

In 1975 and 1976, the Ontario Ministry of the Environment surveyed the asbestos fibre concentrations in the air at a number of locations in the province.[73] The survey report began by stating: "This program was not designed as an air quality survey, but rather it was an attempt to acquire knowledge of the character of the sampled asbestiform fibres from various sources/sites and to get an indication of present asbestos concentrations in Ontario." Air samples were drawn through a Nuclepore filter, the filter was washed ultrasonically in water, and a portion of that water was transferred to a TEM grid for fibre counting. Any fibre with a 3 to 1 length to diameter ratio was considered to be "asbestiform." Among these asbestiform fibres, chrysotile and the amphiboles were identified by morphology, selected area electron diffraction, or energy dispersive x-ray analysis.

The 1975 survey of 7 areas, taking several samples in each area, contained no area with median sample concentrations of fibres longer than 5 microns greater than 0.01 f/cc. In fact, the median count in each area of fibres longer than 5 microns was usually zero or non-detectable. The count of fibres longer than 5 microns in any single sample, if not zero or non-detectable, ranged as high as 0.054 f/cc. The number of fibres of all lengths was considerably greater than the number of fibres longer than 5 microns, but still always less than 1 f/cc. The median mass measurement for each area in 1975 was generally less than 1 ng/m^3, but the highest median was 25.7 ng/m^3. The maximum reading at any location was 184 ng/m^3. It is interesting that the highest mass occurred in Timmins, a city in the heart of Northern Ontario's mining area, where it is at least conceivable that non-asbestos mining activities might cause fibres to become airborne. Kretschmar and Kretschmar have reported various asbestos deposits in the Timmins area.[74] The host rock in non-asbestos mines in the area is tuffaceous, of volcanic origin, which could raise dust that might look like asbestos fibres. Only 14% of the asbestiform fibres in the Timmins data were identified as chrysotile, raising the possibility that some of the remaining fibres, while counted as asbestiform, may have been something other than asbestos.[75]

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[70] Irving J. Selikoff, William J. Nicholson, and Arthur M. Langer, "Asbestos Air Pollution," *Archives of Environmental Health* 25:1 (July 1972): 1-13.

[71] R.W. Lanting and J. den Boeft, *Atmospheric Pollution by Asbestos Fibres*, Report no. G908 (Delft, Holland: Instituut voor Milieuhygiene en Gezondheidstechniek, T.N.O., 1979).

[72] Leonard Bruckman and Robert A. Rubino, "Asbestos: Rationale Behind a Proposed Air Quality Standard," *Journal of the Air Pollution Control Association* 25:12 (December 1975): 1207.

[73] Ontario, Ministry of the Environment, Air Resources Branch, "Asbestos as a Hazardous Contaminant, Progress Report II; Asbestos Ambient Air Monitoring Survey," Report no. ARB-TDA-20-76, Toronto, February 1976.

The Ministry of the Environment's supplementary report, containing the 1976 survey results, concluded by stating:

> The fibre concentrations reported in this survey were consistently an order of magnitude smaller than comparable samples in the previous survey. This may be due to a systematic difference in the analysts' procedures, however, both programs provided internally consistent data. Since the analytical methodology was not standardized at this time, the results should be considered semi-quantitative and used for comparison purposes only. That is, those results should not be taken as a basis for air quality assessment.[76]

This illustrates the difficulties that have been encountered in measuring ambient asbestos levels. The concentration of fibres longer than 5 microns is indeed quite low, with median concentrations of zero or the detection limit in most locations. Once again, the concentration of fibres longer than 5 microns is a small fraction of the concentration of fibres of all lengths, indicating the typical predominance of small fibres in the samples.[77] Recognizing that the optical microscope cannot see thin fibres detected by the electron microscope, we could divide the count of fibres longer than 5 microns by 10, to conclude that there is very little exposure greater than the optical microscope equivalent of 0.001 f/cc. However, the 1976 report discouraged the use of these figures as indicators of asbestos fibre concentrations, because of the great variation between the two sets of measurements and the undeveloped state of the measurement technology at the time. As in the 1975 survey, the mass measurements are correspondingly low, with only one median mass measurement greater than 1 ng/m^3 and the highest mass measurement equal to 36 ng/m^3.

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[74] Ulrich Kretschmar and Dianne Kretschmar, *Talc, Magnesite, and Asbestos Deposits in the Kirkland Lake - Timmins Area, Districts of Timiskaming and Cochrane,* Ontario Geological Survey Open File Report 5391 (Toronto: Ontario Ministry of Natural Resources, 1982).

[75] Ontario, Ministry of the Environment, "Asbestos as a Hazardous Contaminant, Progress Report II," Table 2, p. 26.

[76] Ibid., "Asbestos Air Monitoring Survey: Supplement to Report ARB-TDA-20-76," Report no. ARB-TDA-20S-78, Toronto, 1978, p. 4.

[77] Ibid.

The most recent Ontario data have been collected in a study performed by Dr. Eric J. Chatfield of the Ontario Research Foundation for this Commission.[78] The results of that study are summarized in Table 11.4. This study used the direct preparation of the sample for TEM analysis rather than the indirect method used in the 1975 and 1976 Ontario studies.

The asbestos fibre concentrations found by Chatfield in the rural area near Bracebridge, Ontario, were extremely low. In analyzing 10 samples, Chatfield found only 2 small fibres. In 8 samples, no fibres were counted. Thus, the fibre concentration was reported as less than the detection limit, or 0.0005 f/cc even in the most contaminated sample. In the city of Peterborough, only 4 fibres were found in 3 samples, yielding a maximum concentration of less than 0.001 f/cc.

The other samples were taken adjacent to an expressway ramp in downtown Toronto and in two suburban locations in Mississauga and Oakville. The greatest asbestos fibre concentrations in the study were found in the expressway ramp location, but even here the maximum concentration was only 0.0042 f/cc over 5 microns in length, and the median concentration was less than 0.0033 f/cc. Thus, the maximum concentration was only 10% of the current Ontario Ministry of the Environment Ambient Air Quality Guidelines for Asbestos. The suburban fibre concentrations were still lower.

Chatfield summarized these data as follows:

> The airborne chrysotile concentrations in all locations amounted to a few thousandths of a fibre/mL. No amphibole fibres were detected. The number of chrysotile fibres counted for each measurement was very low, and most of the values were close to the detection limits. Where large mass concentrations were detected, it was usually found that most of the mass was accounted for by one thick fibre.[79]

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[78] Eric J. Chatfield, *Measurement of Asbestos Fibre Concentrations in Ambient Atmospheres*, Royal Commission on Asbestos Study Series, no. 10 (Toronto: Royal Commission on Asbestos, 1983).

[79] Ibid., p. 75.

Table 11.4

Ontario Airborne Asbestos Concentrations*
1982

Location (No. of Samples)	Asbestos Fibre Concentration				Asbestos Fibre Mass	
	All Lengths		Over 5 Microns		All Lengths	
	Median f/cc	Maximum f/cc	Median f/cc	Maximum f/cc	Median ng/m^3	Maximum ng/m^3
Toronto (12) (Expressway Ramp)	0.0034	0.0084	<0.0033	0.0042	0.025	20
Mississauga (8) (Suburb)	<0.0009	0.004	0.0007	<0.002	0.0	0.017
Oakville (13) (Suburb)	0.001	0.0063	<0.0009	0.002	0.0012	8.81
Rural (10) (near Bracebridge)	<0.0004	<0.0005	<0.0004	<0.0005	0.0	0.0079
Peterborough (3) (Small City)	0.0018	0.0019	<0.001	<0.001	0.014	0.24

Notes: *All asbestos is chrysotile.
Fibres counted by TEM following direct preparation method.

SOURCE: The numbers in this table are derived from Dr. Chatfield's raw data. Dr. Chatfield reports confidence intervals rather than individual fibre counts. See Eric J. Chatfield, *Measurement of Asbestos Fibre Concentrations in Ambient Atmospheres,* Royal Commission on Asbestos Study Series, no. 10 (Toronto: Royal Commission on Asbestos, 1983), Tables 8, 9, 10, 11, 12, pp. 77-81.

All of the fibre counts reported in Table 11.4 were taken by electron microscope. Because the electron microscope can detect thin fibres that could not be observed under an optical microscope, the fibre concentration in Table 11.4 could be divided by 10 to approximate roughly the concentration of fibres that could be visible under an optical microscope. After dividing by 10, all concentrations of fibres longer than 5 microns would be well below the 0.001 f/cc level.

Considering all of the above data together, we conclude that asbestos fibre concentrations in the ambient air are extremely low. Counts of fibres longer than 5 microns taken by electron microscope are often less than 0.001 f/cc. If we consider the fibres that would be seen by an optical microscope, it is extremely rare in Ontario to have concentrations greater than 0.001 f/cc. The recent Ontario data suggest that fibre levels are lowest where population density is lowest, although the earlier Ontario data did not reveal this relationship. In Chapter 9 we conclude that the health risks presented to building occupants from exposure to 0.001 optically visible fibres per cubic centimetre is not significant. It follows that the fibre levels discussed in this section present a clearly insignificant health risk. We see no reason to worry about the health effects of the prevalent level of asbestos fibres in the outdoor air in Ontario.

Our conclusion that ambient asbestos fibre concentrations present no health risk is supported by at least one study of persons exposed to ambient asbestos, but not exposed occupationally. Siemiatycki studied the health of female residents of two Quebec asbestos mining towns who were not employed in the mines, but were regularly exposed to more than 1,000 ng/m^3 of asbestos in the air of the town, perhaps two orders of magnitude greater than the ambient concentration in Ontario. This may be considered as the equivalent of 0.03 f/cc measured optically. (See Chapter 9, Section C.) No significant excess of respiratory cancer was detected among these women.[80]

The 1980 *Report of the Working Group on Air Quality in the Toronto Subway System* found asbestos concentrations in the air of the subway system, measured by TEM, ranging from levels below 0.002 f/cc to 0.3 f/cc.[81] This study indicated that the Ministry of the Environment air quality guideline of 0.04 f/cc was exceeded in 13 out of 52 cases. This comparison seems to misuse the air quality guideline which was derived assuming exposure for 24 hours per day, 7 days per week. Subway patrons would rarely be exposed more than one hour per day. Operators would rarely be exposed more than 40 hours per week. In a follow-up study, undertaken after asbestos brake linings on the subway cars were replaced with non-asbestos substitutes, the Ontario Research Foundation found that none of the samples taken exceeded the Ministry of the Environment standard.[82] Chatfield reported some of the follow-up data in one of his studies for this Commission.[83]

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[80] Jack Siemiatycki, "Health Effects on the General Population (Mortality in the General Population in Asbestos Mining Areas)," in *Proceedings of the World Symposium on Asbestos*, Montreal, Quebec: 25-27 May 1982 (Montreal, P.Q.: Canadian Asbestos Information Centre [1983]), p. 342.

[81] Ontario, Ministry of the Environment, Ministry of Labour, and Toronto Transit Commission, *The Report of the Working Group on Air Quality in the Toronto Subway System*, Report ARB-TDA-12-80 (Toronto: Ontario Ministry of the Environment, March 1980), p. 31.

The widespread occurrence of very low concentrations of asbestos fibres in the ambient air is confirmed by studies looking for asbestos fibres caught in human lung tissue. Uncoated asbestos fibres and asbestos bodies, which are fibres coated with iron and protein, have been discovered in the lungs of urban and rural dwellers who have not been occupationally or para-occupationally exposed to asbestos. For instance, Churg and Warnock discovered uncoated asbestos fibres and asbestos bodies in the lungs of 21 randomly selected subjects of routine autopsies who had fewer than 100 asbestos bodies per gram of lung.[84] They confirmed the lack of occupational exposure in 20 out of these 21 urban residents. This led them to associate this concentration of asbestos fibres and bodies with environmental exposure to asbestos. Using a variety of techniques, Churg and Warnock further identified that 80% of the fibres were chrysotile and the remainder amphiboles. The majority of both types of fibres were less than 5 microns long. This confirms that most residents of urban areas do inhale and retain some asbestos fibres as a matter of course.

C.3 Sources of Airborne Asbestos Fibres

A number of sources may be responsible for emitting asbestos into the ambient air. Asbestos may be introduced into the air by natural mechanisms, such as wind erosion of asbestos-bearing rock and soil. In Ontario, there are natural outcroppings of asbestos, but they are in regions which are not densely populated and are not intensely farmed. Natural contamination in Ontario would constitute at most a very minor source of fibre release in rural environments and a negligible source of release in major urban areas. On the other hand, it is possible that mining activities in areas where the natural rock formations contain some asbestos may release some fibres into the air. This is suggested as the cause of elevated fibre levels found in Timmins, Ontario, in the 1976 Ministry of the Environment study discussed above.[85] In addition, the use of asbestos-containing mine tailings on the surface of gravel roads could cause considerable asbestos fibre release if there were a substantial asbestos content in the tailings. We are not aware that such tailings are currently used in Ontario and therefore see no need to regulate their use. In the United States, the use of such tailings for road coverings is prohibited.[86] If in the future the Ministry of the Environment should learn of the use of asbestos-containing tailings on gravel roads, it should conduct tests to determine the ambient fibre levels that result. If a significant problem is detected at that time, regulations prohibiting the use of asbestos-containing tailings on road surfaces should be developed.

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[82] Ontario, Ministry of the Environment, Ministry of Labour, and Toronto Transit Commission, *The 1980 Follow-Up Report on Air Quality in the Toronto Subway System*, Report ARB-TDA-63-80 (Toronto: Ontario Ministry of the Environment, December 1980), Table 4.1, pp. 11-12.

[83] Chatfield, *Measurement of Asbestos Fibre Concentrations in Ambient Atmospheres*, Table 17, p. 94.

[84] Andrew M. Churg and Martha L. Warnock, "Numbers of Asbestos Bodies in Urban Patients with Lung Cancer and Gastrointestinal Cancer and in Matched Controls," *Chest* 76 (2 August 1979): 143-149.

One significant asbestos source in urban areas may be the renovation or demolition of buildings containing asbestos insulation if safe practices are not followed. This is discussed in Chapters 9 and 10.

It has been suggested that airborne asbestos fibres might result from the erosion of asphalt highway paving which contains asbestos. An asphalt mixture containing 2 to 3% chrysotile has been used as an overlay on roadways in North America. Asbestos was included because it rendered the roadway more impermeable to water and thus less likely to crack or deteriorate. Two Ontario municipalities reportedly used asbestos in the past as a binding agent with asphalt on their roads. We are not aware of any municipalities in Ontario currently using asbestos in highway coatings.[87]

We have seen no evidence that asbestos fibres become airborne during the wearing of roadway surfaces. Because the fibres are mixed in asphalt it seems unlikely that they could become airborne in significant quantities. We conclude in Chapter 9 that asbestos contained in liquids such as paint and roofing compounds do not present health hazards to users.

Because it is unlikely that asbestos fibres can be released from asphalt highway surfaces in significant quantities, we are not recommending that any action be taken to limit the use of asbestos-containing asphaltic highway coatings. The problem of controlling worker exposure during bag opening and mixing of asbestos with the asphalt coating is dealt with in Chapter 10. (See Recommendation 10.18.)

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[85] Ontario, Ministry of the Environment, "Asbestos as a Hazardous Contaminant, Progress Report II," p. 2.

[86] 40 CFR 61.20 et seq.

[87] There is one company in Ontario producing non-asbestiform tremolite for use in highway coatings.

It has been suggested that in an urban setting, wear of automotive brake linings may be a significant source of asbestos air pollution. Although there is strong evidence that asbestos fibres can be converted to non-fibrous substances during lining wear, the exact nature and levels of the emissions of wear products from brake linings is still a matter of ongoing debate. For example, Jacko and DuCharme suggested that the high temperatures experienced during vehicle braking caused 99.7% of the asbestos in the brake linings to be converted into inert and harmless particles.[88] Similar results were reported in work undertaken by Lynch; Hatch; Hickish and Knight; Anderson et al.; and Williams and Muhlbaier.[89] Williams and Muhlbaier conducted 23 analyses of wear products from disc brakes and 24 from drum brakes. They found that the average asbestos content of the wear products was 0.029%, which means that over 99.9% of the asbestos in the brake pad or shoe was transformed chemically or physically so that it was no longer an identifiable asbestos fibre.[90] Furthermore, they found that less than 1% of all asbestos fibres observed were longer than 5 microns. On the other hand, Alste, Watson, and Bagg concluded that the major effect of braking appears to be the separation of bundles of fibres and reduction of average length without any crystal structure alteration.[91] Jacko has suggested that the wear debris collected by Alste, Watson, and Bagg may have resulted from studying brake linings with poor wear characteristics.[92]

Rohl et al. analyzed the wear debris from brake drums of automobiles and found that in general only 3 to 6% by weight of the debris was recognizable asbestos, implying that 94 to 97% of the debris was some other material.[93] They further determined that such asbestos as was present in the wear debris consisted predominantly of short fibres. About 80% of the asbestos fibres in the debris were shorter than 3,750 Angstroms (0.375 microns) in length so that a very small fraction, perhaps 1%, of these fibres would have been longer than 5 microns.[94] Thus, researchers seem to be in agreement that brake wear produces material that is 6% or less asbestos, and that less than 1% of these asbestos fibres are longer than 5 microns.

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[88] Michael G. Jacko and Robert T. DuCharme, "Brake Emissions: Emission Measurements from Brake and Clutch Linings from Selected Mobile Sources," EPA Report 68-04-0020, NTIS PB-222-372 (Southfield, Michigan: Bendix Research Labs, March 1973).

[89] Jeremiah R. Lynch, "Brake Lining Decomposition Products," *Journal of the Air Pollution Control Association* 18 (1968): 824–826; D. Hatch, "Possible Alternatives to Asbestos as a Friction Material," *Annals of Occupational Hygiene* 13:1 (January 1970): 25-29; D.E. Hickish and K.L. Knight, "Exposure to Asbestos During Brake Maintenance," *Annals of Occupational Hygiene* 13:1 (January 1970): 17-21; A.E. Anderson et al., "Asbestos Emissions from Brake Dynamometer Tests," SAE Paper 730549 presented at Society of Automotive Engineers Meeting, Detroit, Michigan, 14–16 May 1973; and Ronald L. Williams and Jean L. Muhlbaier, "Asbestos Brake Emissions," *Environmental Research* 29 (1982): 70–82.

[90] Williams and Muhlbaier, "Asbestos Brake Emissions," p. 70.

[91] J. Alste, D. Watson, and J. Bagg, "Airborne Asbestos in the Vicinity of a Freeway," *Atmospheric Environment* 10:8 (1976): 583–589.

[92] Michael G. Jacko, "Physical and Chemical Changes of Organic Disc Pads in Service," *Wear* 46 (1978): 163.

[93] Arthur N. Rohl et al., "Asbestos Exposure During Brake Lining Maintenance and Repair," *Environmental Research* 12 (1976): 125.

Anderson et al. calculated brake dust emission levels in the atmosphere assuming that over 99% of the asbestos was converted to harmless dust.[95] Utilizing an air pollution dispersion model, they estimated that urban asbestos levels from brake lining wear might be 0.07 ng/m^3. Williams and Muhlbaier estimated that brake lining dust emissions might cause urban airborne asbestos concentrations of 0.063 ng/m^3, less than 10% of observed urban asbestos levels reported above.[96] If the conversion rate were lower, so that, for example, 6% of brake wear debris consisted of asbestos fibres, then the simulated airborne asbestos levels attributable to brakes would be at least 6 times greater than the simulated levels arrived at in these studies. In this case, brake wear debris might account for half or more of the airborne asbestos fibres in urban areas. The weight of this evidence is that it is likely that automotive brakes cause only a fraction of the airborne urban asbestos detected, although it is possible that the fraction may be greater than one-half. In any event, we have found urban airborne asbestos levels to be extremely low.

There are a large number of uses of asbestos, many of which could contribute to ambient airborne asbestos levels. We conclude that ambient airborne asbestos concentrations arise from a number of sources, including automotive brakes, work on asbestos-containing buildings, fugitive emissions from asbestos manufacturing plants, and numerous other small sources. No single source is clearly dominant.

C.4 Current Regulations

We have established above that ambient airborne asbestos fibre concentrations are generally so low that they do not present a significant health risk to the general public, even in urban areas. We have identified several probable contributors to airborne asbestos concentrations, but no one source seems to account for a large proportion of airborne asbestos, and the relative importance of various sources is as yet quite uncertain. In this situation, the purpose of establishing ambient air quality standards cannot be to protect the public from generally prevailing asbestos levels, since the risks from these levels are not significant. Furthermore, prevailing asbestos levels cannot generally be attributed to a particular source so that violations could not often lead to regulatory action. However, ambient air quality standards as well as emission standards could be used as a basis for regulating the emissions from individual sources that might otherwise contribute substantially to airborne concentrations. There follows a review of existing regulations of airborne asbestos levels. These regulations focus upon controlling emissions from individual sources.

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[94] Ibid., Table 2, p. 117.

[95] Anderson et al., "Asbestos Emissions from Brake Dynamometer Tests."

[96] Williams and Muhlbaier, "Asbestos Brake Emissions," p. 81.

(a) The United States

In March 1971, the Environmental Protection Agency of the United States named asbestos as a hazardous air pollutant. In April 1973, the National Emission Standard for Asbestos was passed under section 112 of the *Clean Air Act, 1970*, providing that there must be no "visible emissions" to the outside air from asbestos mills, various manufacturing and fabricating establishments using asbestos, asbestos spraying operations, and friable asbestos-containing demolition and renovation sites.[97] In lieu of meeting the "no visible emissions" standard, specific air-cleaning procedures may be followed. The use of asbestos tailings for the surfacing of roadways is expressly prohibited.

The transportation of commercial asbestos is governed by the Asbestos Regulation of the U.S. Department of Transportation.[98] Commercial asbestos must be transported in rigid, leak-tight packaging, non-rigid packaging in enclosed vehicles, or dust- and silt-proof, non-rigid packages which are placed in fibreboard or wooden boxes or which are palletized and unitized (unless transported by private carrier by highway). The container must be marked as containing asbestos.

(b) The United Kingdom

There is little specific statutory control over asbestos in the ambient air in the United Kingdom. Several laws provide the authority to regulate pollution, including asbestos release, but this authority has not been exercised by specific asbestos regulations. The U.K. Advisory Committee on Asbestos recommended more specific control over the emission of asbestos dust from the workplace and recommended that imported raw asbestos be packed in totally enclosed metal-clad containers and that the transportation of asbestos products liable to produce dust be performed so as to prevent the escape of any dust.[99] These recommendations have not yet been acted on. However, it seems that draft regulations dealing with emissions from the workplace and with the transportation of new asbestos, among other substances, are now under consideration in the United Kingdom.[100]

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[97] 40 CFR 61.20 et seq.

[98] 49 CFR 173.1090, 44 FR 47937, 16 August 1979.

[99] U.K., Advisory Committee on Asbestos, *Asbestos — Volume I: Final Report of the Advisory Committee*, Recommendations 32, 35, and 36, pp. 89, 91.

[100] Telephone communication between Mr. Stanley King and Royal Commission on Asbestos Staff, 29 June 1983.

(c) Canada — Federal Provisions

In Canada, the federal *Clean Air Act* authorizes monitoring and research with respect to air pollutants.[101] In addition, the Asbestos Mining and Milling National Emission Standards Regulations, passed under the *Clean Air Act*, establish an emission standard applicable to asbestos mills or mines of 2 f/cc (over 5 microns and with a length to width ratio of 3 to 1 or more).[102] Mine and mill operators are required to keep records of emission measurements and to provide them to administrative authorities when requested.[103] The regulations specifically provide that they do not affect more stringent provincial emission standards.[104] The more general Ambient Air Quality Objectives Order No. 1, passed under the same Act, does not set an objective for asbestos, but does provide that the presence of suspended particulate matter be considered "acceptable" only at levels of 60 to 70 milligrams per cubic metre (mg/m^3) (annual) or 0 to 120 mg/m^3 (24 hours) and "desirable" at a level of 0 to 60 mg/m^3 (annual).[105] These concentrations, being in milligrams, are a million times greater than asbestos concentrations, in nanograms, normally found in outdoor air and are therefore irrelevant for asbestos control. Regulations are currently being developed to govern establishments manufacturing asbestos-containing products.[106]

Finally, the Canada Dangerous Substances Regulations,[107] promulgated under the *Canada Labour Code*,[108] provide that every employer must ensure that any "dangerous substance" that may be carried into the air be confined as closely as is "reasonably practicable" to its source.[109]

(d) Other Provinces

The provinces are entitled to regulate the intra-provincial emission of pollutants, as related to their jurisdiction over property and civil rights, and matters of a local and private nature.[110] All provinces have enacted legislation governing public health, most of which provides for control of "nuisances" by local health authorities. "Nuisance" is typically defined to include any activities that may be harmful to the health of the general public and consequently can be extended to the presence of asbestos.

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[101] S.C. 1970-71-72, c. 47.

[102] *Clean Air Act*, Asbestos Mining and Milling National Emission Standards Regulations, CRC, Vol. IV, c. 405, s. 4(1), p. 2875.

[103] Ibid., s. 6(1).

[104] Ibid., s. 3.

[105] *Clean Air Act*, Ambient Air Quality Objectives Order 1, CRC, Vol. IV, c. 403, p. 2869.

[106] Glasbeek, *A Survey of Asbestos Policies in Canada with Particular Emphasis on Ontario*, p. 42. Confirmed in telephone communication between Mr. R. Capowski, Environment Canada and Royal Commission on Asbestos Staff, 17 May 1983.

[107] *Canada Labour Code*, Canada Dangerous Substances Regulations, CRC, Vol. X, c. 997, p. 7749.

[108] R.S.C. 1970, c. L-1.

[109] Canada Dangerous Substances Regulations, s. 9.

[110] *Constitution Act, 1867* (formerly named the *British North America Act, 1867*), ss. 92(13) and 92(16).

Most provinces have enacted legislation governing the emission of pollutants or contaminants in the air. With the exceptions of Quebec, British Columbia, and Ontario, provinces have not promulgated specific emission standards for asbestos, although standards governing particulates and asphalt plants may be indirectly applicable.

Quebec's Regulation Respecting the Quality of the Environment restricts emissions from establishments processing asbestos to 2 f/cc (over 5 microns in length and with a length to width ratio of at least 3 to 1).[111] When loading asbestos, the same standard, or in the alternative a standard of "no visible emissions" from a distance of 2 metres, is prescribed.

British Columbia's Pollution Control Objectives for the Mining, Smelting and Related Industries provide that asbestos emissions should be restricted to 2 f/cc (greater than 5 microns in length and with a length to width ratio of at least 3 to 1). These objectives are enforced as conditions on permits assigned to emitters. As well, British Columbia has adopted an ambient air guideline for asbestos of 0.04 f/cc; this guideline does not have the force of law.[112]

(e) Ontario

The *Public Health Act* of Ontario, like similar legislation enacted in other provinces, allows local public health authorities to regulate "nuisances" which, by definition, include "[a]ny condition . . . that is or may become injurious or dangerous to health . . . ."[113]

The *Environmental Protection Act* of Ontario prohibits emissions of "contaminants" in excess of prescribed levels[114] and emissions which may impair the quality of the environment, harm any person, or adversely affect the health of any person.[115] The General—Air Pollution Regulations promulgated under that Act establish prescribed emissions levels.[116] While they do not specifically apply to asbestos, the general visible emissions standard included therein may be applicable.

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[111] O.C. 3845-80 (1981) 113 *Gazette Officielle* II 87.

[112] Telephone communication between Mr. W.H. Weldon, Technical Services Division, Waste Management Branch, British Columbia Ministry of the Environment and Royal Commission on Asbestos Staff, 12 July 1983.

[113] R.S.O. 1980, c. 409, s. 115.

[114] R.S.O. 1980, c. 141, s. 13.

[115] Ibid., s. 13(1).

[116] R.R.O. 1980, Reg. 308.

The Ministry of the Environment has developed a 30-minute average emission standard for asbestos of 5 micrograms per cubic metre at the closest point of impingement. In addition, the Ministry has adopted a 24-hour ambient air quality objective of 0.04 f/cc over 5 microns in length, discussed earlier in this section.[117]

C.5 Discussion and Recommendations

There are two elements to standard-setting for ambient air quality: ambient air quality objectives can be established and emission standards set. Air quality objectives state the acceptable level for a particular pollutant in the ambient air. This level must be established after considering evidence on the health and nuisance problems associated with particular levels of a contaminant in the ambient air. Air quality objectives are not directly enforceable unless it is possible to show that a particular emitter is causing the ambient air objective to be exceeded. The primary usefulness of an ambient air quality objective is that it may identify air quality problems.

Emission standards are directly enforceable in that they relate to emissions from individual sources. In setting an emission standard, it is necessary to consider how the relevant air quality objective can be met. With a common contaminant, like asbestos, the emission standard must be set low enough so that the various sources of the contaminant collectively do not cause the air quality objective to be exceeded. A number of factors must be taken into account, including the physics and chemistry of air transport of the substance. A large element of judgement is unavoidable.

We cannot fully endorse or reject Ontario's existing ambient air quality objective of 0.04 f/cc longer than 5 microns because of the continuing lack of agreement in the scientific community on how environmental measurements should be taken. Today, different analysts use different methods of evaluating environmental air samples, with substantially different results. Chatfield has shown that the indirect preparation method, even when performed in the same laboratory by the same technicians, yields both more fibres and more mass than does the direct preparation method.[118]

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[117] Ontario, Ministry of the Environment, Air Resources Branch, "List of Tentative Standards, Guidelines and Provisional Guidelines for Air Contaminants," January 1982.

[118] Chatfield, *Measurement of Asbestos Fibre Concentrations in Ambient Atmospheres*, pp. 83–94.

With the development of an internationally accepted measurement method, better data on the size distribution of asbestos fibres in the ambient air should emerge. Such data would permit an improved understanding of the relationship between TEM (or other) data and the optical microscope data which have been gathered in the work setting and on which the health evidence in relation to the inhalation of asbestos is based. It would then be sensible to reassess Ontario's ambient air quality objective for asbestos.

We do, however, believe that the 0.04 f/cc ambient air quality guideline now in force in Ontario provides a reasonable interim standard. The 0.04 f/cc guideline, combined with the ability of the TEM to detect more thin fibres than the optical microscope, provides more than adequate protection to the public during this interim period. We note that West Germany is contemplating an ambient air quality guideline for asbestos of 1 fibre per litre, or 0.001 f/cc, longer than 5 microns measured by TEM.[119] Our conclusion on the health risks associated with asbestos in the ambient air indicates that the risk reduction, if any, afforded by such a guideline over one that was, say, one order of magnitude greater, is trivial or insignificant.

We believe that the current Ontario ambient air quality criterion for asbestos should remain in force, pending the development of an internationally accepted measurement method. We accordingly recommend that:

11.5 The Ministry of the Environment, in co-operation with the Ministry of Labour, should financially support the development of internationally accepted methods for ambient air quality measurement and adopt such measurement methods when they have been adopted by such international bodies as the International Organization for Standardization.

Once a method for measuring ambient asbestos concentrations has been adopted internationally, and is approved by the Ontario Ministry of the Environment, the Ministry should review the 0.04 f/cc asbestos guideline. In selecting the appropriate level, we believe it would be appropriate for the Ministry of the Environment to consider our conclusion that the health risks presented to the public by protracted exposure to the optical microscope equivalent of 0.001 f/cc longer than 5 microns would not pose a significant health risk to the general public. This assessment of risk from exposure to 0.001 f/cc longer than 5 microns is based on extensive, rather than short-term, exposure. Where the typical period of exposure is brief, considerably higher levels might safely be allowed.

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[119] Ibid., Table 16, p. 92.

The emission standard for asbestos now in place in Ontario is 5 micrograms per cubic metre, at the closest point of impingement. This standard is not related to concentrations of fibres longer than 5 microns, as is the current Ontario ambient air quality guideline. Consistent with our conclusion that long, thin asbestos fibres pose the greatest health risk, we believe that the technique for measuring asbestos-containing emissions should focus on fibres longer than 5 microns. As discussed in Chapters 7 and 9, the relationship between mass measurements of fibres of all sizes and concentrations of long, thin fibres is variable. Mass measurements are generally determined by counting fibres by TEM and determining mass based on this fibre count. The mass determination is unnecessary for the purpose of detecting fibres longer than 5 microns. Long fibres can be counted more accurately if the operator is not distracted by counting the far more numerous short fibres.

As well, fibre counting usually proceeds by examining portions of a microscope grid and counting the fibres found therein until some specified number of fibres has been reached. Accordingly, if short fibres are included in the count, counting will be abandoned when relatively few long fibres have been found; when short fibres are not included, a larger portion of the grid is examined and a greater number of long fibres found.

We conclude that the 5 microgram per cubic metre emission standard should be replaced with a standard specified in terms of fibres per cubic centimetre longer than 5 microns. This emission standard should at present be set so as to be consistent with the current Ontario ambient air quality guideline. We accordingly recommend that:

11.6 The Ministry of the Environment should revise the emission standard for asbestos to specify concentrations of fibres longer than 5 microns. This standard should be consistent with the existing ambient air quality criterion for asbestos.

The emission standard for asbestos should be reassesed when a new ambient air quality criterion for asbestos is set. As stated above, this should follow the establishment of an internationally accepted measurement method.

D. Waste Disposal

D.1 Current Practices and Exposures

The increase in asbestos waste generated by the removal of asbestos-containing insulation from buildings has heightened the problems of asbestos waste disposal in Ontario. Until 1979, the waste management policy of the Ministry of the Environment was based on the premise that asbestos waste was derived from fixed industrial sources, for example, friction products manufacturing. However, with the implementation of programmes to remove asbestos-containing insulation from schools and other buildings, the waste disposal problem has been highlighted and has changed in character. The disposal of asbestos waste from asbestos-containing insulation removal projects, as well as waste from fixed industrial sources, must be subject to a regulatory framework. This is to protect waste disposal workers themselves.

When asbestos is deposited in a landfill site, degradation of the asbestos will depend on the nature of the site, including soil conditions. The report of the U.K. Advisory Committee on Asbestos explained: "If conditions are very acidic, which may occur from other chemical waste disposal, then chrysotile asbestos may well be degraded, although amphibole asbestos varieties will be virtually unaffected by changes in the acidity or alkalinity of disposal sites. Composite wastes such as asbestos cement may be subject to chemical changes in the soil which would remove the bonding agents, leaving the asbestos fibre unaffected. Similarly, resinated asbestos materials would be subject to bacterial and chemical attack which would free the fibrous component which would remain virtually undegraded."[120] However, even if the asbestos does not degrade, the potential for fibres to migrate through soil is minimal,[121] unless the sites are dug up or are situated on ". . . fissured, fractured or jointed strata leading to aquifers and where water-filled sites are in hydraulic continuity with surface and groundwater resources . . . ."[122] Accordingly, asbestos fibres deposited in appropriate landfill sites and properly overlaid are unlikely to contaminate air or water resources.

A study undertaken by Her Majesty's Factory Inspector (HMFI) in the United Kingdom between 1976 and 1977 investigated levels of asbestos in the ambient air around waste disposal sites. Atmospheric dust sampling was carried out at 79 different waste disposal sites. Of the 236 samples taken, 25% were from the breathing zone of the workers, 65% from the background, and 10% from outside the site perimeter. Only 2 non-crocidolite samples exceeded the occupational hygiene standard then in force for non-crocidolite asbestos fibres of 2 f/cc, with readings of 2.3 and 2.4 f/cc. The vast majority of samples collected ranged from 0.01 to 0.2 f/cc. Two samples which contained crocidolite exceeded the 0.2 f/cc standard then in force for crocidolite, with readings of 0.6 and 8.6 f/cc. The reading of 8.6 f/cc may be explained by the fact that split bags were observed spilling their contents on the site where the sample was taken. This suggests that negligent work practices when disposing of asbestos waste lead to elevated airborne asbestos fibre levels at disposal sites. None of the samples taken by the HMFI beyond the site boundaries yielded figures in excess of the hygiene standard.[123]

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[120] U.K., Advisory Committee on Asbestos, *Asbestos — Volume 1: Final Report of the Advisory Committee*, paragraph 46, p. 37.

[121] Wallace H. Fuller, *Movement of Selected Metals, Asbestos and Cyanide in Soil: Applications to Waste Disposal Problems*, EPA-600/2-77-020 (Cincinnati, Ohio: U.S. Environmental Protection Agency, 1977), p. 4.

[122] U.K., Department of the Environment, *Waste Management Paper No. 18, Asbestos Wastes: A Technical Memorandum on Arisings and Disposal Including a Code of Practice* (London: Her Majesty's Stationery Office, 1979), p. 11.

The above data indicate the need for appropriate work practices for the disposal of asbestos waste. Such work practices will protect both disposal workers and members of the public dwelling near disposal sites.

Since asbestos is not highly degradable, workers involved in future developments of asbestos-containing landfill sites may be at a risk from exposure to airborne asbestos fibres. Accordingly, it is important that records be kept regarding the locations of asbestos waste, so that those workers may be protected.

Operators of waste disposal sites in Ontario are not required to accept asbestos waste. We have been informed that in a number of areas in the province, municipal waste site operators have refused to accept waste materials generated from asbestos-containing insulation removal projects. Removal contractors have accordingly had to rely on private disposal sites. However, some private site operators are also reluctant to permit asbestos to be dumped at their sites. As a result, removal contractors are forced to stockpile waste material near the sites of removal operations. The Commission has learned of occasions when asbestos scraps were transported to farmers' fields and dumped there for minimal fees. In other cases, asbestos waste has been indiscriminately placed in open pits.[124] Accordingly, it is necessary that a system be devised that ensures the availability of adequate and safe asbestos dumping grounds.

It appears that the Ministry of the Environment has not been sufficiently diligent in monitoring asbestos waste disposal, in stark contrast to the extensive precautions undertaken to protect workers during removal of asbestos from buildings. Dissatisfaction with the Ministry of the Environment's monitoring of waste disposal is widespread in the asbestos removal industry. In discussions with our staff, contractors have urged that if downstream treatment of asbestos waste is not rigorously handled by the Ministry of the Environment, then following a rigorous regime to prevent asbestos exposure during asbestos removal may be pointless.

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[123] U.K., Advisory Committee on Asbestos, *Asbestos — Volume 1: Final Report of the Advisory Committee*, paragraphs G5-G6, p. 43.

[124] The staff of the Royal Commission on Asbestos has had discussions with removal contractors, government safety inspectors, and waste disposal site operators on these matters.

The Ministry of the Evironment has largely left to the Ministry of Labour the monitoring of known asbestos waste sources to ensure conscientious preparation and handling of asbestos waste materials. However, the Construction Health and Safety Branch of the Ministry of Labour has told the staff of this Commission that little attention is paid by its field personnel to the waste transfer and disposal stages of asbestos removal operations. Moreover, because the Ministry of Labour does not require notification of contracts valued at less than $50,000, many waste-generating operations are completely unregulated.[125]

The monitoring of dump sites by the Ministry of the Environment has been criticized. For example, in Ottawa, *The Citizen* published a series of reports on asbestos waste disposal at the Nepean dump in May 1981. In that case, for three months, ". . . drums [of asbestos], instead of being buried in a special area of the dump, went to the normal landfill site where they were run over by compacting machines or tossed down a 10-metre-deep gully, causing them to burst open and spilling their contents."[126] Until *The Citizen* warned the site operator, he was unaware of special handling procedures for asbestos. Furthermore, although the Ministry of the Environment officer was aware that asbestos was being deposited in the Nepean dump, he was not acquainted with the specific techniques that should be employed by dumps when they dispose of asbestos waste. We have been told that similar incidents have occurred at dump sites elsewhere in Ontario.[127]

D.2 Current Regulations: The United States, The United Kingdom, and Ontario

(a) The United States

Procedures for the disposal of asbestos waste generated from manufacturing, demolition, renovation, and spraying operations in the United States are outlined in the Environmental Protection Agency's National Emission Standard for Asbestos.[128] Disposal operations must meet the "no visible emissions" standard or, in the alternative, compliance with the regulation may be achieved by waste being wetted, sealed in a leak-tight container, and labelled;[129] or by processing asbestos waste into non-friable forms.[130] In the case of waste from asbestos mills, the last alternative is not acceptable. The owner or operator of the waste-producing source is responsible for compliance with the waste disposal regulation.

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[125] Construction Projects Regulation, R.R.O. 1980, Reg. 691, s. 4(1)(a), under the *Occupational Health and Safety Act*, R.S.O. 1980, c. 321.

[126] See *The Citizen* (Ottawa), 5, 6, 7 May 1981.

[127] The staff of the Royal Commission on Asbestos has had discussions with asbestos removal contractors and dump site operators.

[128] 40 CFR 61.20 et seq.; 40 CFR 61.22(j).

[129] 40 CFR 61.22(j)(3)(i).

Active waste disposal sites must exhibit no visible emissions or, in the alternative, asbestos waste must be covered daily with non-asbestos-containing material.[131] Warning signs must be displayed on the waste disposal site property and at all entrances.

Inactive waste disposal sites that once received asbestos must meet the "no visible emission" standard or cover the asbestos waste with non-asbestos materials.[132] The warning sign provisions applicable to active sites also govern inactive sites.

In all cases, the owner or operator of the existing source must provide to the EPA information concerning the type of disposal site used, the name and location of the site, and the name of the site operator.[133]

(b) The United Kingdom

Under the *Control of Pollution Act, 1974,* local authorities are given the power to establish licensing schemes for the control of waste,[134] and power to designate and regulate "special waste," meaning those wastes which may be dangerous or difficult to dispose of.[135] The Control of Pollution (Special) Waste Regulation of 1980 passed under this statute includes asbestos in the definition of "special waste."[136] Consignment notes are required to be used by producers, carriers, and disposers of waste. Labelling and disposal requirements are outlined as well.

(c) Ontario

Waste disposal in Ontario is currently governed by Part V of the *Environmental Protection Act* through the General—Waste Management Regulation promulgated under that Act.[137]

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[130] 40 CFR 61.22(j)(3)(ii).

[131] Fifteen centimeters of non-asbestos-containing material or a resinous or petroleum-based dust suppression agent [40 CFR 61.25(c)(1) and (2)].

[132] Fifteen centimeters of non-asbestos-containing material and a vegetation cover or 60 centimeters of compacted non-asbestos-containing material or a resinous or petroleum-based dust suppression agent [40 CFR 61.22(k)(5)].

[133] 40 CFR 61.24(c).

[134] 22-23 Eliz. II, c. 40, ss. 1 and 2.

[135] Ibid., s. 17.

[136] S.I. 1980/1709, s. 2(1) and Schedule I, Part I.

[137] See R.S.O. 1980, c. 141 and R.R.O. 1980, Reg. 309 thereunder.

Part V of the *Environmental Protection Act* requires that all "waste disposal sites" and "waste management systems" be certified.[138] The definition of "waste management systems" includes collection, transportation, and handling of waste; hence certified approval is required of haulers of waste. Use of uncertified facilities is prohibited.[139]

In March 1983, the Ministry of the Environment filed a Regulation amending the General—Waste Management Regulation with regard to asbestos waste.[140] This 1983 Regulation defines "asbestos waste" as solid or liquid waste that contains asbestos in more than a trivial amount or proportion.[141] Procedures for handling asbestos waste are prescribed by the 1983 Regulation.

Under the 1983 Regulation, no person may cause or permit asbestos waste to leave the location at which it was generated, except for the purpose of transporting it to a waste disposal site, the operator of which has agreed to accept it.[142] The asbestos waste must be placed in rigid, impermeable, sealed containers. If the container is a cardboard box, the waste must be sealed in a 6 mil polyethylene bag placed within the box.[143] If the asbestos waste is transported in bulk, it must be transported by means of a waste management system operating under a certificate of approval specifically authorizing the transportation of asbestos waste in bulk.[144] A specific caution against asbestos hazards is required to be placed on containers and vehicles.[145] Drivers transporting asbestos waste must be trained in asbestos waste management and must transport the waste as directly as is practicable to the waste disposal site.[146] Vehicles carrying asbestos waste in cardboard boxes must be enclosed. In all other cases, vehicles that are not enclosed must be covered with a suitable tarpaulin or net.[147]

Once deposited at a disposal site, asbestos waste must be covered forthwith with at least 125 centimetres of garbage or cover material.[148] Every person handling asbestos waste or its containers, supervising the unloading of asbestos waste in bulk, or cleaning asbestos waste residues from containers, vehicles, or equipment must wear protective clothing and personal respiratory equipment.[149] There are provisions for proper handling of protective clothing.[150] Finally, all persons involved in the transportation, handling, or management of asbestos waste must take all necessary precautions to prevent asbestos waste from becoming airborne.[151]

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[138] R.S.O. 1980, c. 141, s. 27.

[139] R.S.O. 1980, c. 141, ss. 39 and 40.

[140] O. Reg. 175/83.

[141] O. Reg. 175/83, s. 1(1).

[142] Ibid., s. 14(1).

[143] Ibid., ss. 14(1) and 14(2).

[144] Ibid., s. 14(1).

[145] Ibid., s. 14(5).

[146] Ibid., s. 14(6).

[147] Ibid.

[148] Ibid., s. 14(11).

[149] Ibid., s. 14(12).

D.3 Recommendations

We endorse the steps recently taken by the Ministry of the Environment in regulating the handling of asbestos waste. We expect that proper enforcement of its 1983 Regulation will greatly improve asbestos waste handling in this province. Dissatisfaction with asbestos waste handling in Ontario hitherto expressed by members of the asbestos removal industry should accordingly be alleviated.

The 1983 Regulation on asbestos waste appears to apply to all sources of asbestos waste. We believe that it is not realistic to expect that this Regulation would be effectively enforced in dealing with sources of small amounts of asbestos waste, such as those generated by brake lining repair work. We accordingly recommend that:

11.7 The Ministry of the Environment should amend the 1983 General—Waste Management Regulation made under the Environmental Protection Act so as to provide that the sections regulating asbestos waste apply only to asbestos waste generated by asbestos removal operations and by asbestos product manufacturing.

It is necessary that the Ministry of the Environment be notified prior to the transportation of asbestos waste, so that the regulation of asbestos waste management can be properly enforced. We accordingly recommend that:

11.8 The Ministry of the Environment should amend the 1983 General—Waste Management Regulation made under the Environmental Protection Act so as to require that the Ministry be notified, prior to the transporting of asbestos waste, of the quantity of the waste, the nature and destination of the waste, the type of packing involved, and any other pertinent details.

We note above that there is concern over the availability of disposal sites for asbestos waste in Ontario, because disposal site operators sometimes refuse to accept such waste. Moreover, we have noted that when asbestos is deposited in disposal sites without records being kept as to its precise location, workers on future disposal site developments are placed at risk. While these sites cannot be redeveloped for 25 years after their being used for waste disposal without the special permission of the Minister of the Environment,[152] available evidence suggests that asbestos waste may not degrade over such a period. Accordingly, we conclude that asbestos should be deposited either in designated, recorded areas of existing disposal sites or, if necessary, in special asbestos disposal sites, and that the Ministry of the Environment should take steps to ensure that there are adequate facilities for the disposal of asbestos waste. We therefore recommend that:

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[150] Ibid., ss. 14(13) and 14(14).

[151] Ibid., s. 14(15).

11.9 The Ministry of the Environment should amend the 1983 General—Waste Management Regulation made under the Environmental Protection Act so as to require that precise records be kept as to the location of deposited asbestos waste. The content and storage of these records should be specified by the Ministry.

We further recommend that:

11.10 The Ministry of the Environment should determine which disposal site operators are prepared to accept asbestos waste. If this determination indicates that there are not adequate disposal sites for receiving asbestos waste in the province, the Ministry of the Environment should either establish special asbestos disposal sites or consider requiring certain disposal site operators to accept asbestos waste.

We are also concerned about disposal sites where asbestos was dumped in the past. For example, Johns-Manville has reported to the Commission that there are 1.6 million tons of landfill material present at its Scarborough plant area, of which up to 5% is asbestos. Johns-Manville has plans to redevelop this area.[153]

Workers involved in the eventual redevelopment of disposal sites which contain significant quantities of asbestos should be protected. It is advisable that policy be developed as to the restrictions on redevelopment that should apply to such sites. We accordingly recommend that:

11.11 The Ministry of the Environment should develop policies regarding the redevelopment of waste sites known to contain substantial quantities of asbestos.

Finally, there is the problem of asbestos having been disposed of in unidentified sites in the past. Where it is reasonably practicable, it would be desirable to identify the location of substantial quantities of asbestos waste so that the policies developed under Recommendation 11.11 can be applied to them. We therefore recommend that:

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[152] R.S.O. 1980, c. 141, s. 45.

[153] Letter from Johns-Manville Canada Inc. to the Royal Commission on Asbestos, 31 August 1981.

11.12 The Ministry of the Environment should, where reasonably practicable, endeavour to identify sites in which substantial quantities of asbestos have been deposited.

Volume

3

Ontario

Report of The Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario

Volume Three

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Part VI

Compensating Victims: Asbestos and Its Implications

Chapter 12 Determining Eligibility for Asbestos-Related Disease Compensation

A. Introduction

Our terms of reference require us to "review the present basis for Workmen's Compensation Board awards as they relate to occupational health matters affecting workers exposed to asbestos, including any special programmes dealing with the rehabilitation of such workers." We were not asked to review the entire framework for workers' compensation. The Government of Ontario assigned this task to Professor Paul C. Weiler on January 30, 1980, some four months before we were appointed. While our own inquiry was in progress, Professor Weiler published, in November 1980, an initial report entitled *Reshaping Workers' Compensation for Ontario.*[1] This was followed in June 1981 by a *White Paper* setting out in the form of a legislative exposure draft important proposals for change in the Ontario workers' compensation system.[2] These proposals remained in the realm of discussion when we held our formal hearings on workers' compensation in the summer of 1982, some five months later than originally planned. The delay was occasioned in part to accommodate interested parties, whose own time resources had been pressed by the need to react to the *White Paper.* By the fall of 1982 and after the conclusion of our hearings, the acceptance of the *White Paper* proposals remained a matter for speculation. A slightly revised legislative draft of these proposals[3] was placed before the Standing Committee on Resources Development of the Ontario Legislature, whose report was to be expected in 1983. No government bill would be introduced before the Standing Committee's report was received. Whatever their disposition, the *White Paper* proposals would be joined in 1983 by a second Weiler report which, among other things, would address general issues in the realm of industrial disease compensation.[4]

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[1] Paul C. Weiler, *Reshaping Workers' Compensation for Ontario*, a report submitted to Robert G. Elgie, M.D., Minister of Labour ([Toronto: Ontario Ministry of Labour], November 1980).

[2] Ontario, Ministry of Labour, *White Paper on the Workers' Compensation Act* [Toronto: Ontario Ministry of Labour, 1981].

Thus, fundamental changes in the basic framework of workers' compensation have been in the air throughout our inquiry. We have viewed this as an opportunity rather than a hindrance and indeed maintained contact with Professor Weiler during our work. The fact of the matter is that we could not take any fundamental change in the framework of compensation as a given because all such changes awaited decision as we conducted our own deliberations.

In this setting, it was our own best judgement to approach the issues posed by asbestos-related compensation and rehabilitation in a manner that would be sensitive to: (i) the possibility that there may be little change in the compensation framework governed by the current *Workers' Compensation Act*[5] and that such changes as we might wish to recommend should therefore be confined to asbestos-specific (or disease-specific) amendments to the current legislation; (ii) the possibility that a framework basically similar to the legislative exposure draft set out in the *White Paper* might be incorporated into a government bill and that such changes as we might wish to recommend should address the content of this bill; and (iii) the possibility that a quite novel approach to compensation contemplated by Professor Weiler in his second report could eventually divorce some or all industrial diseases and accidents from the workers' compensation framework in favour of a general compensation framework.

Of these three possibilities, the first two are less readily compartmentalized than the third. The practical result is that some of our recommendations simultaneously invite amendments to the current legislation and endorse the changes proposed in the legislative exposure draft contained in the *White Paper*. In the chapters that follow, we shall indicate where this is the case. We shall also indicate where our recommendations invite amendments to whatever statute might provide the legal framework for workers' compensation in Ontario.

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[3] Ontario, Standing Committee on Resources Development, *Hearings on Reshaping Workers' Compensation in Ontario* and on the *White Paper on the Workers' Compensation Act*, Exhibit 11a, "Changes in the Draft Act since June 25, 1981," filed by the Ontario Ministry of Labour, 8 September 1982.

[4] Paul C. Weiler, *Protecting the Worker from Disability: Challenges for the Eighties*, a report submitted to Russell H. Ramsay, Minister of Labour ([Toronto: Ontario Ministry of Labour], April 1983).

[5] The *Workmen's Compensation Act* was renamed the *Workers' Compensation Act* by the *Workers' Compensation Amendment Act, 1982*, R.S.O. 1982, c. 61, assented to December 21, 1982. By this same Act the Workmen's Compensation Board was similarly renamed the Workers' Compensation Board. Generally, references in the text of this Report use the term "Workers' " whereas footnote references indicate the term in use at the date of reference.

No issues before this Commission have proved more sensitive than those posed by workers' compensation. The informal phases of our hearings elicited from victims and survivors numerous submissions and presentations, many of them touchingly spontaneous. As a royal commission, we also pursued the matter of workers' compensation through sworn testimony under the procedures prescribed by the *Public Inquiries Act*. To help ensure an open ventilation of all sides of the question, we granted legal standing and public funds to the Asbestos Victims of Ontario. We commissioned a special study, entitled *Workers' Compensation and Asbestos in Ontario*, by an internationally recognized expert, Professor Peter S. Barth of the University of Connecticut.[6] This study was released in February of 1982 and figured prominently in the formal hearings we held during the following summer. It stimulated written commentaries from the Injured Workers' Consultants, the Workers' Compensation Board (WCB), and the Executive Director of the Board's Medical Services Division.[7] We ourselves commissioned two reviews by outside experts, Professor Terence G. Ison of Osgoode Hall Law School and Dr. Adam S. Little, former Chairman of the British Columbia Workers' Compensation Board.[8]

Notwithstanding the specific focus of our terms of reference on asbestos-related disease, the issues we must address are multi-faceted and have wide-ranging implications. We choose to address these issues as follows. First, there is the complicated question of determining eligibility for asbestos-related disease compensation through adjudication, eligibility rules, and appeals. This is the subject of the present chapter. Next, there are a number of procedural and substantive issues that arise from the handling of asbestos-related disease claims. These will be addressed in Chapter 13. Finally, we shall address in Chapter 14 issues of rehabilitation, outreach, and prevention as they relate to workers' compensation.

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[6] Peter S. Barth, *Workers' Compensation and Asbestos in Ontario*, Royal Commission on Asbestos Study Series, no. 2 (Toronto: Royal Commission on Asbestos, 1982).

[7] Nick McCombie, "Response to Peter Barth's *Workers' Compensation and Asbestos in Ontario* by Injured Workers' Consultants," Toronto, April 1982; The Workmen's Compensation Board, "Comments on the Study and Review Papers on Workmen's Compensation and Asbestos in Ontario," Toronto, 6 August 1982; and William J. McCracken, "Comments and Critique on a Study Prepared by Peter S. Barth, *Workers' Compensation and Asbestos in Ontario*," Toronto, 6 August 1982. (Mimeographed.)

[8] Reviews of studies prepared for the Royal Commission on Asbestos: Terence G. Ison, "*Workers' Compensation and Asbestos in Ontario:* A Commentary on the Report of Peter S. Barth," Toronto, April 1982; and Adam S. Little, "Critique re: *Workers' Compensation and Asbestos in Ontario*," Victoria, B.C., 1982. (Mimeographed.)

B. The Determination of Eligibility

B.1 The Current Setting

The *Workers' Compensation Act,* in section 122(1), states that "Where an employee suffers from an industrial disease and is thereby disabled or his death is caused by an industrial disease and the disease is due to the nature of any employment in which he was engaged," he is entitled to compensation.[9] The words of section 122(1) reflect a legislative amendment passed in 1973 that is of importance to asbestos-related disease compensation. At that time, a long-standing requirement that to be eligible for compensation, industrial disease victims must be disabled from earning full wages was abolished.[10] This permitted individuals in early stages of asbestosis who could nonetheless continue their employment to receive partial compensation.

The *Workers' Compensation Act* gives the Board wide authority to compensate industrial disease victims. It leaves the Board broad discretion to determine what is an industrial disease; whether or not an employee suffers from the disease; the extent to which the employee is thereby disabled; whether or not the employee's death was caused by the disease; and whether the disease was due to the nature of the employment. While the Act contains provisions that structure the Board's discretion, these feature less prominently, by the Board's own choice, in asbestos-related disease than in certain other industrial diseases. As we acknowledge later in this chapter, the Board has devised non-statutory means of structuring its discretion, but these have not been made widely known.

A generally acknowledged canon of public administration holds that where law ends, discretion begins. An historically hallowed dictum of constitutionalism holds in contrast that "where law ends, tyranny begins."[11] Which of these dicta applies to the current setting of asbestos-related disease compensation in Ontario? The first dictum applies as a matter of fact. The second applies as a matter of perception. Regretfully, our inquiry compels us to say that the Board is widely perceived to apply its discretion in a manner so capricious as to suggest to the beholder that the second dictum has elements of validity.

On the one hand, we have been most favourably impressed by the competence, sincerity, and resourcefulness of the Board officials and consultants who appeared before us. These are medical specialists and professional public administrators whose good intentions are beyond question and whose devoted actions over the years have applied the Board's discretion in a manner that places Ontario at the forefront of industrial disease compensation policy. It is a matter of fact that the Ontario Board was the first workers' compensation agency in North America to compensate asbestos-related lung cancer in the absence of clinical asbestosis and the leader in compensating laryngeal and gastrointestinal cancer among asbestos workers.

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[9] R.S.O. 1980, c. 539, s. 122(1).

[10] *The Workmen's Compensation Amendment Act, 1973 (No. 2),* S.O. 1973, c. 173, s. 9(1).

[11] The words "Where law ends, tyranny begins," are inscribed in the facade of the U.S. Department of Justice Building in Washington. For a seminal treatment of the relationship between law and discretion, see Kenneth Culp Davis, *Discretionary Justice: A Preliminary Inquiry* (Baton Rouge: Louisiana State University Press, 1969).

On the other hand, we cannot dismiss the disparaging remarks of victims, survivors, and persons experienced in presenting appeal cases to the Board. "To me," a lawyer who has long dealt with the Board told us, "it's just a jungle."[12] For his part, Professor Ison, in his review of the Barth study, expressed the opinion that the Board, in applying its discretion, has hopelessly entangled questions of law, policy, medical fact, and non-medical fact.[13] In this light we take yet other comments made to this Commission, however much some may have bordered on the vitriolic, as anything but crank views. We take them instead as the healthy reaction that citizens of a democratic society should have to the perception of tyranny.

Such a perception need not and should not be valid. As the noted legal scholar Kenneth Culp Davis has observed, tyranny will not begin where law ends if discretion has been adequately structured and confined.[14] The Board's problem must therefore lie in the extent to which discretion in determining the compensation eligibility of disease victims has lacked structure and confinement either in appearance or in fact. The approaches that have been available to the Board are statutory presumption, guidelines, and case-by-case adjudication. Let us examine how these approaches have been used.

B.2 Statutory Presumption

This approach to the determination of eligibility for disease compensation is made available by section 122(9) of the *Workers' Compensation Act* which states:

> If the employee at or immediately before the date of the disablement was *employed* in any *process* mentioned in the *second*

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[12] Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Submission by Mr. Daniel Ublansky on behalf of the Energy and Chemical Workers Union, Volume no. 47(B), 5 July 1982, p. 116.

[13] Ison, "*Workers' Compensation and Asbestos in Ontario:* A Commentary on the Report of Peter S. Barth," p. 14 and *passim*.

[14] Davis, *Discretionary Justice: A Preliminary Inquiry*. This is the basic argument of Professor Davis' book.

> *column* of *Schedule 3* and the *disease* contracted is the *disease* in the *first column* of the Schedule set opposite to the description of the *process*, the disease shall be deemed to have been due to the nature of that employment unless the contrary is proved . . . . (Emphasis added.)[15]

Section 122(9) creates a statutory presumption in favour of any claimant whose disease and employment satisfy the content of Schedule 3 by stating that this individual's disease "shall be deemed to have been due to the nature of that employment unless the contrary is proved." It structures the discretion of the Board in the matter of eligibility for disease compensation by making it clear that the stipulations of Schedule 3, when satisfied, generate in favour of the claimant a rebuttable presumption, that is, a presumption the burden of whose refutation must be borne by others.

The Schedule 3 to which section 122(9) refers is as old as workers' compensation in Ontario. The original statute of 1914 included a schedule which associated six diseases with six industrial processes.[16] In 1926, Schedule 3 was amended to provide, first, for silicosis in relation to mining and, second, for pneumoconiosis as it related to the processes of "quarrying, cutting, crushing, grinding, or polishing of stone, or grinding or polishing of metal."[17] The addition of pneumoconiosis to Schedule 3 made asbestosis a compensable disease. In 1942, the first asbestosis claim was compensated.[18]

Until 1947, it was a requirement that a disease be listed in Schedule 3 in order to be eligible for compensation. On March 31, 1947, the Act was amended by enacting the forerunner to the present section 1(1)(n) which defines industrial disease as including not only diseases mentioned in Schedule 3 but "any other disease peculiar to or characteristic of a particular industrial process, trade or occupation."[19] This amendment greatly liberalized industrial disease compensation policy in Ontario. It also vested the Board with the broad discretion it possesses to the present day. Schedule 3, meantime, remained available as a means of structuring the Board's discretion and indeed was made more readily available than heretofore by an amendment, also enacted in 1947, which transferred the approval of future changes in Schedule 3 from the Legislature to the Lieutenant Governor in Council.[20] Henceforth the Board need only secure an order-in-council to change Schedule 3. But for reasons cited later in this chapter, the Board chose not to take advantage of this more flexible means of structuring its enhanced discretion.

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[15] R.S.O. 1980, c. 539, s. 122(9).

[16] *The Workmen's Compensation Act*, S.O. 1914, c. 25.

[17] See S.O. 1926, c. 42; and Amendment to Regulation 94, 1 June 1926.

[18] The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, p. 1.

[19] S.O. 1947, c. 119, s. 1(1)(h)(ii).

[20] S.O. 1950, c. 89, s. 10. See also, Barth, *Workers' Compensation and Asbestos in Ontario*, p. 5.3.

The last episode in the story of Schedule 3 as it concerns asbestos-related disease was written in 1955. As of January 1, 1956, the processes that had hitherto been listed in the second column of the Schedule opposite pneumoconiosis were listed opposite silicosis. Pneumoconiosis reappeared in the first column (the disease column) as "the pneumoconioses other than silicosis."[21] Opposite these words the second column (the process column) was left blank. The statutory presumption triggered by section 122(9) was thereby rendered inoperable with respect to asbestosis claimants and remains so to the present day. Even before this apparently anomalous situation came about, the Board had decided to veer away from statutory presumption as a means of structuring its discretion.

B.3 Guidelines

In 1947, at the very time when it became possible to change Schedule 3 merely by having recourse to an order-in-council, the Board ". . . concluded that with the proliferation of substances and conditions . . . it would be impossible to constantly keep adjusting Schedule 3."[22] Apparently, what led the Board to eschew statutory presumption was the issue of work-relatedness, the very issue that makes the compensation of industrial disease more challenging than the compensation of industrial accidents. In the words of the Board's current Vice-Chairman of Administration and General Manager in testimony before us: ". . . the evidence was that the absolute relationship between industrial process and those conditions was not such that you could justify automatic presumptions. . . ."[23] As an alternative to Schedule 3, the Board might be able to structure its discretion through guidelines. In the meantime, the Board would adjudicate disease claims from case to case, approaching each claim as if it were entirely new. "Historically," Professor Barth noted, "this has been the [Board's] approach when very few claims for a specific disease had materialized or were expected."[24] According to Professor Barth, the initial guidelines to be applied to the processing of industrial disease claims emerged in 1949 (lung cancer in relation to coal tar); 1956 (lung cancer in relation to arsenic-

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[21] O. Reg. 230/55 under *The Workmen's Compensation Act*, as printed in *The Ontario Gazette*, 17 December 1955, p. 2370.

[22] RCA Transcript, Evidence of Mr. Alan G. MacDonald, 11 August 1982, Volume no. 55, p. 13.

[23] Ibid.

[24] Barth, *Workers' Compensation and Asbestos in Ontario*, p. 5.2.

smelting); 1959 (lung and sinus cancers in relation to nickel refining); a
1969 (lung and sinus cancers in relation to sintering).[25]

Ongoing attention to the development of guidelines is not appar
until the mid-1970s, and coincides with the appointment of the Board's c
rent Executive Director of the Medical Services Division, Dr. William
McCracken, who assumed his post in 1975. As Dr. McCracken told
when he testified at our hearings: "It was my opinion that wherever p
sible . . . guidelines of various types should . . . be developed, because t
would be of real assistance in the handing down of uniform decisions.'
The words of this witness capture the essential purpose of guidelines (or f
that matter statutory presumptions): to serve the goal of horizontal equi
that is, the similar treatment of similarly situated individuals.

The credentials of the individuals, whether staff or consultants, w
have been involved in developing Board guidelines are impeccable, a
their analysis of the controversial scientific issues posed by asbestos-relat
diseases has been studied and thorough. However, the manner in whi
Board guidelines have been processed and publicized is not commensura
with the importance of the equity goal they are meant to serve. T
development of the current asbestos-related disease guidelines is covered
length in Professor Barth's study and need not be repeated here. On t
basis of our hearings, we choose to offer observations rather than detail
description. For the purposes of this chapter, the guidelines to which
allude are those for lung cancer (approved by the Board on April 13, 197
mesothelioma (approved April 13, 1976); gastrointestinal cancer (approv
October 7, 1976); and laryngeal cancer (approved in provisional form M
4, 1978). The text of these guidelines is provided in the Appendix to t
chapter. We defer to Chapter 13 the question, raised by Professor Barth,
whether or not an asbestosis guideline is in existence, as this is an importa
illustration of the difficulties that have beset the processing of individu
claims. And we defer to Chapter 14 discussion of the Board's pre-disea
Asbestos Fibre Dust Effect guideline because it is related to rehabilitatio
Our observations with respect to the mesothelioma and asbestos-relat
cancer guidelines can be distilled into the following points:

(i) The unstructured nature of the Board's guideline-setting process
borne out by evident disagreement between the Board's Vice-Chairman
Administration and the Executive Director of the Board's Medical Servi

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[25] Ibid., p. 5.5. Professor Barth also noted on pp. 5.4 and 5.6 a few guidelines develop
between 1956 and 1972 whose range of coverage was limited to a single firm. T
Executive Director of the Board's Medical Services Division has referred to these as dir
tives rather than guidelines. See RCA Transcript, Evidence of Dr. William J. McCrack
20 July 1982, Volume no. 53, pp. 34-35.

[26] RCA Transcript, Evidence of Dr. William J. McCracken, 20 July 1982, Volume no. 5
p. 23.

Division regarding details of that process. In testimony, the former sustained Professor Barth's contention[27] that the Management Committee plays the initiating role by forming a subcommittee to devise guidelines,[28] and that the Management Committee ultimately conveys guidelines to the Corporate Board for its approval.[29] The latter took issue with Professor Barth and spoke instead of the central role of an *ad hoc* committee struck at the initiative of the Executive Directors of the Medical Services and Claims Services Divisions[30] and of the role of these officials in subsequently transmitting guidelines directly to the Corporate Board.[31] Which view is correct is less important than what this evident disagreement tells us about the informality of the guideline-setting process.[32]

(ii) Professor Barth's description of guideline-setting did not mention the Board's Joint Consultative Committee. This Committee, created by a 1973 amendment to *The Workmen's Compensation Act*,[33] is composed of Lieutenant Governor in Council appointees, "representative of labour, management and the public,"[34] who are advisory to the Corporate Board. In making no reference to this Committee, Professor Barth correctly conveyed the fact that it was not active in the development of the mesothelioma and asbestos-related cancer guidelines. However, we have been informed by the Board that when the laryngeal cancer guideline related to asbestos and nickel exposure was released in May of 1978, the Committee expressed a desire to become involved in future guidelines.[35] The Committee's subsequent input into the development of non-asbestos disease guidelines is testimony to the Corporate Board's willingness to take a step away from what has been an entirely closed and internal guideline-setting process, but by way of reaction rather than on its own initiative.

(iii) The content of the mesothelioma and asbestos-related cancer guidelines is indeed, as Professor Barth pointed out, at variance with some of the expert medical consultants' findings.[36] Bearing in mind that the guidelines are intended to structure the Board's discretion in adjudicating claims, su variance from expert findings is not inherently a defect, because t guidelines by their very purpose are at once more and less than scienti statements. For the Board to make a guideline somewhat more stringe than expert findings indicate could be taken as expressing the pragma judgement that closer case-by-case scrutiny of a larger volume of clai remains advisable in the face of uncertainty. Conversely, for the Board promulgate a guideline with terms less stringent than indicated by exp findings would express the equally pragmatic judgement that the volume case-by-case scrutiny which would result from adhering to these findings not warranted and that relatively automatic compensation of what wou otherwise be agonizingly debatable claims is to be preferred. Either judg ment involves considerations which, because they affect the relative volu of cases that must receive close scrutiny, are matters of adjudicative poli If this seems reasonable, however, the content of the Corporate Boar lung cancer guideline offers a rather arresting instance of two pragma judgements that run in opposite directions. Here the Corporate Boa chose to be more stringent than its expert by adopting a minimum exposu duration of 10 years where he had none (this increases the need for case-b case scrutiny); and then less stringent than the same expert by stipulating minimum latency period of 10 years where he had 15 (this decreases t need for case-by-case scrutiny). We take this as indicative of a less th systematic approach to guideline content at the level of the Corpora Board.

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[27] Barth, *Workers' Compensation and Asbestos in Ontario*, pp. 5.12, 5.13.

[28] RCA Transcript, Evidence of Mr. Alan G. MacDonald, 11 August 1982, Volume no. 55, pp. 45–46.

[29] Ibid., pp. 47–48.

[30] RCA Transcript, Evidence of Dr. William J. McCracken, 20 July 1982, Volume no. 53, pp. 28–29.

[31] Ibid., pp. 29–30.

[32] The description of the process given to us by the Executive Director of the Claims Services Division is basically in line with that offered by the Executive Director of the Medical Services Division. See RCA Transcript, Evidence of Mr. John F. McDonald, 13 July 1982, Volume no. 48, pp. 158–159.

[33] *The Workmen's Compensation Amendment Act, 1973 (No. 2)*, S.O. 1973, c. 173, s. 8.

[34] R.S.O. 1980, c. 539, s. 71(3)(h).

[35] See "The Joint Consultative Committee on the Workmen's Compensation Board — Comments on its History and Involvement in the Development of Guidelines for the Adjudication of Industrial Diseases," attached to letter from Mr. Alex Joma, Secretary, The Workmen's Compensation Board to the Royal Commission on Asbestos, 23 August 1982.

[36] Barth, *Workers' Compensation and Asbestos in Ontario*, pp. 5.12–5.24.

(iv) Even in the limited realm of asbestos-related disease, the use of guid lines as a means of structuring the Board's discretion has been selecti rather than general. Nothing makes the point more tellingly than t absence of a guideline for compensating the survivors of partially disabl asbestotics. It is a requirement of the *Workers' Compensation Act* tha with the exception of deceased individuals who were in receipt of a 100 permanent disability pension,[37] survivor benefits shall be allowed only if t victim's ". . . death is caused by an industrial disease and the disease is d to the nature of any employment in which he was engaged. . . ."[38] This r quirement, whatever its merits, has posed as acute an adjudicative challen to the Board as it has brought indignant grief to many survivors. W postpone comments on the adequacy of the Board's adjudication t Chapter 13. With respect to the absence of a guideline, however, we no that Professor Barth and the Board witnesses who appeared before us we in full agreement that benefits nonetheless flow to survivors of all partial disabled asbestotics whose deaths from asbestosis, mesothelioma, or a asbestos-related cancer have been verified. Furthermore, the testimony o the Board's Chest Disease Consultant indicated that the Board invariab compensates the survivors of asbestotics who die of *cor pulmonale* (right-sided heart failure arising from pulmonary fibrosis).[39] The non-existence of a guideline which would at least promulgate these established practices as policy speaks tellingly of the Board's piecemeal approach to the structuring of its discretion.

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[37] R.S.O. 1980, c. 539, s. 43(7).

[38] Ibid., s. 122(1). This section is further reinforced by s. 36(1) ("Compensation in case o death"), which begins with the words: "Where death results from an injury. . . ."

(v) It remains clear that the Board intended to use guidelines as a means of structuring its discretion in the adjudication of asbestos-related disease claims. Accordingly, claims that fall within the guidelines will be favourably adjudicated if the facts of the claimant's case satisfy Board officials that the claim falls within the guidelines. It is equally clear, however, that the guidelines were originally intended solely for the internal use of the Board and not as a means of publicizing the fact that the Board was indeed structuring its discretion. Thus, the Board, more particularly the Corporate Board, failed to seize a genuine opportunity to combat perceptions of arbitrariness among its claimants and appellants. In opting, however valid its reasons, to structure its discretion through guidelines rather than the statutory presumptions made available by Schedule 3 of the Act, the Corporate Board either overlooked or ignored the fact that Schedule 3 has always been publicly available. When the Board's guidelines did become public documents in 1979, this was not at the Board's initiative but in response to a recommendation made by the Select Committee on the Ombudsman of the Ontario Legislature.[40] As for the extent to which the guidelines are now publicly disseminated, suffice it to note that they are to be found in a large volume available for $36 at the Ontario Government Bookstore.

If the above observations can be summarized in a single sentence, it is that the Board's recourse to guidelines as a means of structuring its discretion, however useful, has been informal, internal, unsystematic, and piecemeal, and has done nothing to dispel perceptions of Board arbitrariness.

B.4 Case-by-Case Adjudication

The Board's guidelines simplify case-by-case adjudication. Precisely because guidelines structure the Board's discretion, the act of adjudicating claims that ostensibly meet the guidelines is simply a matter of verifying the facts involved. This leaves all remaining claims to fall into one of three categories of case-by-case adjudication: (i) claims that should be covered by guidelines but are not; (ii) claims involving diseases that are subject to guidelines but whose facts fail to meet the guidelines; and (iii) claims whose nature is unfamiliar and may indeed be pathbreaking. Because we have already criticized the Board for its piecemeal approach to guideline-setting, we dismiss the first category by stating that, in our view, it should not exist. This leaves the second and third categories for comment here.

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[39] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, pp. 79–80.

[40] Ontario, *Fifth Report of the Select Committee on the Ombudsman*, Michael N. Davison, MPP, Chairman (Toronto: 9 November 1978), pp. 55, 126, 127, 135.

With respect to claims that fall into the second category, the Board has seen fit to offer a measure of direction concerning the manner in which adjudicators are to approach them. Each of the guidelines for mesothelioma, lung cancer, gastrointestinal cancer, and laryngeal cancer contains the following words:

> Claims which do not meet the guidelines . . . should be individually judged on their own merit having regard to the intensity of exposure and other factors peculiar to the individual case. *The benefit of reasonable doubt applies.* (Emphasis added.)

Two of the four guidelines, that for mesothelioma and that for gastrointestinal cancer, contain an additional sentence that appears between the first and the second of the sentences quoted above:

> Consideration will be given where it seems evident that the [mesothelioma] [gastrointestinal cancer] resulted from occupational exposure to asbestos.

We have no evidence that this additional sentence has any significance. What *is* significant is the directive that is given to adjudicators by the statement "the benefit of reasonable doubt applies." In 1980, the Board revised this statement by removing the word "reasonable," so that its current official version is "the benefit of doubt applies." This revision was made in response to concern expressed by reports of the Select Committee on the Ombudsman[41] and, in the words of the Executive Director of the Board's Medical Services Division, "It was interpreted that [it] would operate more favourably to the worker."[42]

In principle, we can only praise the Board for what it is trying to achieve through a benefit of doubt directive. Eligibility rules such as guidelines serve important purposes but pose their own danger: namely, that adjudicators, merely because a guideline is in place, may approach claims that do not satisfy the guidelines with a negative mind-set. In that the benefit of doubt directive combats this danger, it is laudable.

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[41] Ibid., pp. 37-39, 124-125; and Ontario, *Seventh Report of the Select Committee on the Ombudsman*, Patrick D. Lawlor, MPP, Chairman (Toronto: 17 September 1979), pp. 27-29.

[42] RCA Transcript, Evidence of Dr. William J. McCracken, 20 July 1982, Volume no. 53, p. 67.

How has the benefit of doubt directive been applied in practice? Here mesothelioma claims are to be sharply distinguished from asbestos-related cancer claims. Professor Barth has stated,[43] and our evidence confirms, that mesothelioma claims which do not meet the pertinent guideline are invariably compensated unless there has been no documented occupational exposure to asbestos whatsoever.[44] This may speak, as we shall argue later, for the possibility that the guideline approach to mesothelioma is inadequate; in any event it testifies to the existence of a *positive* mind-set among adjudicators where mesothelioma is concerned. (We duly note in passing that the "adjudicators" involved are an amalgam of lay and medical officials, reflecting the entanglement of policy, fact, and medicine which Professor Ison has criticized and to which we shall return in Chapter 13.)

As Professor Barth pointed out, the situation with respect to asbestos-related cancer claims is different. Here, a number of claims that do not satisfy the guidelines are in fact rejected. We see no anomaly here because we would expect, as a general proposition, that certain claims which do not satisfy the guidelines might indeed be deniable. Our concern lies not in claim denials *per se*, but in the possibility that a negative mind-set would yield relatively automatic denials. Such does not appear to be the case. Professor Barth observed that where a claim does not meet the guidelines, ". . . there still remains a substantial possibility that compensation will be paid."[45] Data supplied to us by the Board with respect to lung cancer claims indicate that, of 52 asbestos-related lung cancer claims which have been allowed, 4 failed to satisfy the guidelines' minimum exposure criterion of 10 years. One successful claim lacked a quantifiable record of exposure; two lacked a quantifiable record of latency. Of 18 lung cancer claims rejected, only 6 involved identified exposure to asbestos, and all of these 6 involved exposure well below the 10 years specified in the guideline. (The mean length of exposure in these 6 claims was 2.68 years, and the one with the longest exposure, 6.29 years, revealed no asbestos bodies in pathological specimens.)[46] We take this as indicative that, at least with respect to guideline-related lung cancer claims in the realm of asbestos, the Board's adjudication has not displayed a negative mind-set.

There remains our third category of case-by-case adjudication; namely, claims that are unfamiliar and are potentially pathbreaking. Here, the wide discretion which the Board enjoys in all matters of industrial disease compensation comes into play, and we would not have it otherwise. In the real world of multiplying workplace hazards and advances in scientific knowledge, the very possibility of eventually structuring discretion in matters of workers' compensation is created by the careful consideration of unusual or historically new claims. We consider such discretion highly desirable, subject only to the confinement generated by benefit of doubt policy and a scrupulously fair appeals procedure. Let us now address eligibility for disease compensation in a prescriptive vein.

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[43] Barth, *Workers' Compensation and Asbestos in Ontario*, p. 5.18.

[44] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, p. 74.

[45] Barth, *Workers' Compensation and Asbestos in Ontario*, p. 5.31.

[46] Ontario, Royal Commission on Asbestos, Exhibit IV-9 [hereafter RCA Exhibit], filed with the Commission prior to the Evidence of Mr. William D. Pearce, 12 August 1982: The Workmen's Compensation Board, Ontario, Inter-Divisional Communication from Dr. Douglas W. Dyer, Chest Disease Specialist to Mr. Alex Joma, Secretary, 10 August 1982.

C. Structuring the Determination of Eligibility for Disease Compensation

C.1 The Need for Institutional Change

Asbestos-specific though our terms of reference may be, our reflections on the matters just discussed have led us to conclude that we would shortchange our assignment if we failed to address subjects that impinge on workers' compensation at the highest level of generality. Accordingly, we begin our prescriptions by considering the make-up of the Corporate Board and the need to overhaul the Board's appeals procedures. In our judgement, Professor Weiler has dealt with these matters cogently and creatively. We therefore make most of our own recommendations by way of either supporting or supplementing his work.

With respect to the make-up of the Corporate Board, we broach a subject that indeed has implications for many compensation matters far outside our terms of reference. But as we proceeded to piece together the story of the Board's development of asbestos-related disease guidelines, our first instinctive reaction led us to question most seriously the adequacy of the present composition of the Corporate Board. How is the informal, internal, unsystematic, and piecemeal quality of the Board's approach to the structuring of its discretion to be explained? In that the avowed purpose of the guidelines was to serve horizontal equity among similarly situated claimants, how could the Corporate Board overlook or ignore the opportunity to give wide publicity to its efforts in this regard? Remarkably, how could this happen during a decade in which the Corporate Board and its agency were being repeatedly and widely excoriated by politicians and the media for acting in a seemingly arbitrary and capricious manner? Our intuitive response is that a substantial part of the answer to these questions must lie in the statutorily prescribed make-up of the Corporate Board as a seven-member body composed entirely of full-time officials (a Chairman, Vice-Chairman of Administration, Vice-Chairman of Appeals, and four Commissioners of Appeals). A membership composition better designed, particularly in the face of outside criticism, to foster an inward-looking, siege mentality could scarcely be devised. By statute and through no fault of its own, the Corporate Board has been deprived of the refreshing perspectives that only part-time directors can bring to the policy deliberations of any major corporation.

We strongly endorse Professor Weiler's argument in favour of a Corporate Board with a majority of outside directors.[47] In so doing, we lay explicit emphasis on the importance of a *majority* to ensure that outsiders will have a guiding influence on corporate deliberations rather than be co-opted as a part-time minority can so often be by full-time officials. Accordingly, we recommend that:

12.1 In line with the legislative exposure draft in the White Paper on the Workers' Compensation Act, the Corporate Board of the Workers' Compensation Board should be composed of a majority of outside directors.

We join Professor Weiler in advocating that the choice of outside directors should bring to the Board ". . . the points of view of labour, management, medicine, vocational rehabilitation, occupational health and safety, and the economics of income maintenance."[48] To this we would add our own view that the choice of outside directors should also be open to a few individuals who would be selected solely on the basis of demonstrated wisdom and common sense in areas totally unrelated to the compensation field. The legislative exposure draft in the *White Paper on the Workers' Compensation Act* envisages a Corporate Board of not more than nine directors, including the Chairman, the Vice-Chairman of Administration, and the Chairman of the Appeals Tribunal. We respect the thrust of Professor Weiler's admonition that the number of directors ". . . be kept small enough to allow the Corporate Board to function as a cohesive deliberative body rather than as a primarily representative institution."[49] However, we view the maximum of nine directors envisaged by the *White Paper* as unduly restrictive. This is not least because, later in this chapter, we find reason to add a fourth official to the echelon that includes the Chairman, the Vice-Chairman of Administration, and the Chairman of the Appeals Tribunal. With a maximum of only nine, the majority of outside directors would dwindle to one. Our own personal experiences in various domains satisfy us that, at no sacrifice in cohesion, a somewhat higher maximum can permit a broader range of representative interests, including those of labour and management, and a more robust majority of outside directors. We therefore recommend that:

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[47] Weiler, *Reshaping Workers' Compensation for Ontario*, pp. 129–133.

[48] Ibid., p. 130.

[49] Ibid.

12.2 Contrary to the legislative exposure draft in the White Paper on the Workers' Compensation Act, the number of Corporate Board members should be fixed at a maximum of twelve.

In the matter of appeals, Professor Weiler's approach merits our enthusiastic endorsement. He has not proposed to change the role either of the Claims Review Branch, which screens every claim whenever it appears that an adverse decision may have to be made, or of the Appeals Adjudicators, who provide the initial level of appeal. We accept Professor Weiler's reasons for confining structural change to the upper echelon of appeals and add two reasons of our own. First, we believe that changes which we propose in Chapter 13 will improve the quality of lower-level claims adjudication with respect to asbestos-related disease. Second, we believe that Professor Weiler's approach to the disclosure of medical reports and opinions will favourably influence the equity of adjudication below the upper echelon of appeals. Translated into the language of the exposure draft legislation in the *White Paper on the Workers' Compensation Act,* this approach stipulates in section 80(1) that:

> *Any* medical report or medical opinion in respect of a worker or deceased worker . . . *from any source whatsoever . . . shall. . . be made available to the worker or claimant for compensation.* (Emphasis added.)

Such statutory language, which is in line with the recommendations of the Commission of Inquiry into the Confidentiality of Health Information (the Krever Commission), should strengthen and broaden the policy concerning access to claim files which the Board promulgated on December 28, 1981. It also appears to us likely that the availability of medical reports and opinions to appellants will, over time, reduce the extent to which, as Professor Ison has observed, issues of policy, non-medical fact, and medicine have been entangled. By way of formally recording our unqualified support of Professor Weiler's approach to the disclosure of medical information, we recommend that:

12.3 The provisions of section 80 of the legislative exposure draft in the White Paper on the Workers' Compensation Act concerning access to medical reports and opinions should be enacted in substantially their present form.

At the upper echelon of appeals, the legislative exposure draft does away with the existing Appeals Boards and with appeals to the Corporate Board in favour of the "Workers' Compensation Appeals Tribunal," which is separate from the WCB and composed of a Chairman, Vice-Chairman, and an unspecified number of members, equally representing employers and workers, all of whom are to be Lieutenant Governor in Council appointees. The decisions of this independent tribunal are binding on the

Corporate Board, save that the Corporate Board may direct the Tribunal to reconsider a matter where a decision of the Tribunal turns upon an interpretation of general law or the policy of the Act.

We are pleased to take due note of the personal support which Mr. Alan G. MacDonald, the Board's Vice-Chairman of Administration, expressed for the proposed appeals structure in his testimony before us.[50] The only reservation which he personally voiced concerned Professor Weiler's proposal that the Chairman of the Appeals Tribunal should sit on the Corporate Board. In Mr. MacDonald's view, this might perpetuate ". . . the public perception . . . that there is an influence by the Board over the appeal group. . . ."[51] Professor Weiler justified his own preference as the most attractive response to the need to ensure an open channel of communication between the policy role of the Board and the adjudicative role of the Tribunal.[52]

We find ourselves most sensitive to the issue of perception, particularly when it can be said that the membership of the Appeals Tribunal Chairman on the Corporate Board might offend natural justice. On the other hand, we find this *open* channel of communication far preferable to the possibility of informal Board-Tribunal intercourse with its own implications for public perception. On balance, wishing to express our overall endorsement of the appeals structure Professor Weiler has proposed, we recommend that:

12.4 The provisions of the legislative exposure draft in the White Paper on the Workers' Compensation Act concerning the structure and procedures of the "Workers' Compensation Appeals Tribunal" should be enacted in substantially their present form.

There is one aspect of Appeals Tribunal procedure which we consider so important that we wish to record a pinpointed endorsement. We refer to section 75(3) of the legislative exposure draft which stipulates:

> Every decision of the Appeals Tribunal, disposing of an appeal, together with its findings and reasons, shall be made in writing.

Supporting provisions in the legislative exposure draft direct that Tribunal decisions shall be promptly communicated in writing to the claimant, employer, or party of record. The *White Paper* stresses the importance of the reasons for Tribunal decisions and stipulates that the Tribunal is to ". . . publish or otherwise make them publicly available."[53]

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[50] RCA Transcript, Evidence of Mr. Alan G. MacDonald, 11 August 1982, Volume no. 55, p. 22.

[51] Ibid., p. 23.

[52] Weiler, *Reshaping Workers' Compensation for Ontario*, p. 117.

In their testimony before us, the Executive Director of the Board's Claims Services Division and the Executive Director of its Medical Services Division each expressed reservations about the publication of decisions.[54] The former official noted that the British Columbia Board may be moving away from its publication practices.[55] The reservations which these two officials harboured are rooted in an honest and proper concern that published decisions could confine matters of disease compensation in a straightjacket of precedents. This concern is well taken, but in our view was grasped fully by Professor Weiler when, in support of his recommendation, he wrote: "I do not advocate practice of rigid adherence to strict precedent . . . . Surely it is high time, though, that Ontario begin to develop a coherent jurisprudence of workers' compensation by using a series of concrete appeals to work out and refine sensible principles and policies."[56] To this we ourselves would only add that openness is the natural enemy of arbitrariness. We therefore recommend that:

12.5 ***The provisions of section 75(3) of the legislative exposure draft in the White Paper on the Workers' Compensation Act concerning the requirement that Appeals Tribunal decisions, including findings and reasons, be made in writing should be enacted into law, together with the supporting provisions requiring communication of decisions to the claimant, employer, or party of record.***

Professor Weiler's final proposal affecting the upper echelon of appeals takes the form of Medical Review Panels (MRPs). Members of MRPs are to be selected from rosters of medical specialists in particular classes of injury or disease. The rosters are to be named by the Lieutenant Governor in Council. Each MRP is to consist of three members drawn from the roster, one named by the claimant, the second by the employer, and the third (the chairman) named by the other two members. An MRP may be constituted at the request of: (i) the Board; (ii) the Appeals Tribunal; or (iii) a claimant or his employer. An application for an MRP by the claimant or his employer, pursuant to section 81(2) of the legislative exposure draft,

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[53] Ontario, Ministry of Labour, *White Paper on the Workers' Compensation Act*, p. 37.

[54] RCA Transcript, Evidence of Mr. John F. McDonald, 14 July 1982, Volume no. 49, p. 113; and RCA Transcript, Evidence of Dr. William J. McCracken, 20 July 1982, Volume no. 53, p. 138.

[55] RCA Transcript, Evideuce of Mr. John F. McDonald, 13 July 1982, Volume no. 48, p. 97.

[56] Weiler, *Reshaping Workers' Compensation for Ontario*, p. 116.

> . . . shall be accompanied by a certificate from a physician stating that in the opinion of the physician there is a *bona fide* medical dispute to be resolved, and such certificate shall include a statement of particulars sufficient to define the questions in dispute.

The decision of an MRP is not necessarily final. Section 86(4) of the *White Paper* legislation empowers the Appeals Tribunal to call for the nomination of a second MRP.

We note all these features with strong approval. We record as well our explicit endorsement of section 83(1) which stipulates that, after an MRP has been appointed,

> . . . the Appeals Tribunal shall prepare a list of medical questions to be determined by the Medical Review Panel and such list shall be accompanied by a review of relevant facts, and a summary of applicable law and policy.

We view this as a most important step towards the disentanglement of medical questions from questions of non-medical fact, law, and policy. We therefore recommend that:

12.6 The provisions of the legislative exposure draft in the White Paper on the Workers' Compensation Act concerning the structure and procedures of Medical Review Panels should be enacted in substantially their present form.

C.2 The Need for Eligibility Rules: The Special Case of Asbestosis and Mesothelioma

The major institutional changes which we have just endorsed will have salutary consequences for the Board's approach to policy development and for the fairness and equity of all compensation decisions made under the authority of its legislation. Let us now address the need for eligibility rules with respect to asbestos-related diseases, beginning with the distinctive situation posed by asbestosis and mesothelioma.

Industrial disease compensation is a field fraught with complexity. With few exceptions, none of which applies to asbestos-related disease, there is a lengthy period between first exposure and the appearance of disease symptoms. Congenital predispositions may make certain individuals peculiarly susceptible to particular diseases. The greatest difficulty posed in the application of a workers' compensation scheme to many diseases is the work-relatedness of these diseases. Here, the determination of a cause-effect relationship between the disease and its associated industrial process is clouded by considerations such as lifestyle and the non-occupational environment. In Professor Weiler's words, "Few diseases are specifically industrial, in the sense that exposure to a particular process or substance is both necessary and sufficient for the employee to contract the disease."[57]

In our considered opinion, asbestosis and mesothelioma are clearly distinguishable from other asbestos-related diseases because they are indeed industry-specific. The evidence that exposure to asbestos is the necessary and sufficient cause of asbestosis is uncontested, and while mesotheliomas are known to occur among individuals who have never had occupational exposure to asbestos, their rarity is such that the possibility that an individual who has been occupationally exposed to asbestos might nonetheless have contracted mesothelioma from a source other than the workplace is extremely remote. Mesothelioma has no association with lifestyle factors such as smoking. As for asbestosis, smoking can affect the progression of the fibrotic process and of its clinical manifestations, but does not initiate the process.

In the face of this robust evidence, assessed by us in Chapter 5, we conclude that asbestosis and mesothelioma should be deemed to be quintessentially industrial diseases whenever there is any evidence of occupational exposure to asbestos. Two important considerations flow from this conclusion. The first is that asbestosis and mesothelioma should be compensable within the framework of workers' compensation for as long as it is not totally subsumed by a general compensation scheme. Where occupational exposure to asbestos is known to have taken place, victims of asbestosis or mesothelioma should be treated in equity as if they were victims of industrial accidents. The second consideration is that this principle should be reflected in the workers' compensation legislation of Ontario.

We have already told how the Board came to veer away from the statutory presumption approach to industrial disease that is embodied in section 122(9) and Schedule 3 of the Act. We consider it most advisable that this approach be reactivated with particular regard to asbestosis and mesothelioma. Especially at a time when Ontario, thanks to Professor Weiler's second report on industrial disease, will have the opportunity to debate seriously the merits of a general compensation policy,[58] we deem it most important not to lose sight of the fact that certain industrial diseases pose no significant questions of work-relatedness. A statutory presumption approach to such diseases provides a visible and easily understood line of demarcation. Because an eligibility rule registered in Schedule 3 requires Lieutenant Governor in Council approval, it acquires a formality that yields to claimants or potential claimants maximum certainty that they can qualify for compensation. Again, policy with respect to an industrial disease is most clearly and widely communicated when it is written into a Schedule that accompanies every copy of the legislation. The higher profile accorded to an industrial disease listed in Schedule 3 may assist outreach efforts that seek to encourage potential claimants to come forward.

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[57] Ibid., p. 139.

[58] Weiler, *Protecting the Worker from Disability: Challenges for the Eighties*, chap. 3.

With respect to the testimony we have received from Board officials, we note that the Executive Director of the Medical Services Division had no knowledge of any Corporate Board deliberations concerning the appropriateness of Schedule 3 for asbestosis and mesothelioma,[59] and that the Vice-Chairman of Administration, under examination by us, agreed that the Board's current approach to mesothelioma in effect coincides with what would be achieved by listing this disease in Schedule 3.[60] In light of all the considerations outlined above, we recommend that:

12.7 The Workers' Compensation Board, at the earliest opportunity, should advise the Lieutenant Governor in Council to amend Schedule 3 of the Workers' Compensation Act by inserting in the first column (the disease column) the words: "Asbestosis and Mesothelioma"; and in the second column (the process column) the words: "Any process involving the use of asbestos."

It is apparent from the testimony of the Executive Director of the Board's Medical Services Division that each disease currently listed in Schedule 3 is directly related to the workplace in the sense that the industrial process involved is necessary and sufficient for the employee to contract the disease.[61] In the interest of horizontal equity among claimants for disease compensation, we believe that Schedule 3 should be reviewed to ensure that this is indeed the case with respect not only to diseases that are currently listed but diseases that meet this specification. We therefore recommend that:

12.8 The Workers' Compensation Board should review Schedule 3 of the Workers' Compensation Act to ensure that it incorporates those diseases whose associated industrial process is necessary and sufficient to cause the disease.

Our designation of asbestosis and mesothelioma as asbestos-specific diseases leads to two additional considerations with respect to section 122(9) and Schedule 3. The first is a matter of statutory language. In referring to Schedule 3, section 122(9) opens with the words, "If the employee *at or immediately before* the date of the disablement was employed in any process mentioned in the second column of Schedule 3 . . . ." (Emphasis added.) This language denies the benefit of Schedule 3 to claimants who were employed in the designated process at an earlier time in their employment history. We note with strong approval that the legislative exposure draft in the *White Paper on the Workers' Compensation Act* proposes to remedy what, given the long latency of asbestos-related disease, is evidently deficient statutory language. Section 8(10) of the legislative exposure draft substitutes for the opening words of section 122(9), "If the worker *at or before* the date of disablement was employed in any process mentioned in the second column of Schedule 3 . . . ." We recommend that:

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[59] RCA Transcript, Evidence of Dr. William J. McCracken, 20 July 1982, Volume no. 53, pp. 48–51.

[60] RCA Transcript, Evidence of Mr. Alan G. MacDonald, 11 August 1982, Volume no. 55, pp. 40–41.

[61] RCA Transcript, Evidence of Dr. William J. McCracken, 20 July 1982, Volume no. 53, pp. 49–53.

12.9 In line with the legislative exposure draft in the White Paper on the Workers' Compensation Act, the provisions of Schedule 3 should be applicable to workers who have engaged in a scheduled industrial process at any time in their employment history.

Our final consideration with respect to section 122(9) concerns the nature of the presumption it generates in favour of a claimant whose disease is covered by Schedule 3. The wording of section 122(9) triggers the presumption if the claimant has the disease named in the disease column of Schedule 3 and was employed in the process designated in the process column of this Schedule. The presumption does not extend to the accuracy of the claimant's disease diagnosis or to whether the claimant's employer engaged in the designated process, but applies once these two matters of fact have been ascertained. It is a rebuttable presumption; that is, it generates in favour of the claimant a presumption the burden of whose refutation must be borne by the employer.

We have considered the submission of the Ontario Federation of Labour that the statutory presumption in favour of the claimant should be irrebuttable, meaning that the validity of a claim which satisfied Schedule 3 could not be challenged.[62] In considering this submission, we have been sensitive to the importance attached by Professor Weiler to a Board relationship with employees and employers that is symmetrical. A particular reason why Professor Weiler favoured symmetry lies in his espousal of stricter experience rating for individual firms. Reserving to Chapter 14 our views on the appropriateness of experience rating in the realm of industrial disease, we join Professor Weiler to the extent that the current Act, by virtue of sections 122(3) and 122(4), gives an employer the right to contend that the claimant's disease was not contracted in his employ. The exercise of this right does not challenge the validity of the compensation claim; it simply raises the question of whether any part of the cost of the claim should be charged to the employer.

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[62] Ontario Federation of Labour, Written submission to the Royal Commission on Asbestos, #78, October 1981, pp. 5–11.

Our concern is over the appropriateness of any challenge to the validity of a compensation claim in situations where the claimant's disease and the employment process are both in line with the specifications of Schedule 3. In his study, Professor Barth reported that "Employer appeals or dissatisfaction were rather rare, and were limited largely to denials that exposures to asbestos occurred in their employ. These charges did not attack the legitimacy of the claim."[63] Indeed, Professor Barth and Board officials agree that the existing guidelines, as currently administered, create *de facto* an irrebuttable presumption in favour of the employee. Nonetheless, the possibility of a rebuttal exists and to our knowledge is currently being used by an employer, no longer in the asbestos business in Ontario, against claims granted while it was still in operation.[64]

When a disease is so directly related to industrial exposure that it qualifies for Schedule 3, we declare ourselves particularly sensitive to the position of the claimant. The long latency of industrial disease normally adds to the time required to process disease, as distinct from accident, claims. Workers' compensation exists to spare victims the uncertainties of the tort liability system. The relative certainty, to say nothing of the dignity, of the survivors of an industrial disease claimant can be badly undermined by an employer's attempt to rebut the validity of that claim. In this light, we conclude that the presumption in favour of a claimant who satisfies Schedule 3 should be irrebuttable, and accordingly recommend that:

12.10 Section 122(9) of the Workers' Compensation Act should be amended so as to stipulate an irrebuttable presumption in favour of the claimant.

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[63] Barth, *Workers' Compensation and Asbestos in Ontario*, pp. 9.21–9.22.

[64] On July 7, 1980, Bendix Automotive of Canada Limited wrote to the Workmen's Compensation Board advising of its intent to appeal the allowance of two mesothelioma claims. Bendix stated that "at the hearing the Company would have the right to introduce independent medical evidence referable to the empirical and medical justification for the diagnosis of mesothelioma and the establishment of a causal connection." Letter from Mr. T. D. Warrington, Vice-Chairman of Appeals, The Workmen's Compensation Board to the Royal Commission on Asbestos, 20 October 1982. However, as of July 1983, the Board had not received any further indication from Bendix that it was proceeding with the appeals, and benefits were continuing in both claims. Letter from Mr. Alex Joma, Secretary, Workers' Compensation Board to the Royal Commission on Asbestos, 25 July 1983.

C.3 The Need for Eligibility Rules: The Case of Asbestos-Related Carcinomas

Asbestos-related carcinomas constitute a case different from asbestosis and mesothelioma. Such cancers all pose the dilemma associated with the vast run of industrial diseases: the issue of work-relatedness. The incidence of lung, gastrointestinal, and laryngeal cancer is widespread. The contribution of asbestos exposure to these cancers is entangled with the contribution of occupational exposure to other hazardous substances, with environmental factors, and with lifestyle.

If a general compensation scheme, even along lines more modest than those envisaged in Professor Weiler's second report,[65] were to be adopted in Ontario, the most cruel of the problems posed by the issue of work-relatedness would be alleviated. The often tenuous relationship between a newly acquired disease and a job held long ago would no longer have all-or-nothing consequences for compensation. The discrepancy between the large number of disease cases which, as we shall see in Chapter 14, experts believe are likely to be work-related and the small number of claims actually granted by a workers' compensation scheme would recede as a source of concern. The need for outreach programmes which seek, with all their inherent limitations, to identify individuals who may have grounds for seeking workers' compensation would be reduced.

A general compensation scheme is a social policy innovation that can draw support from the above considerations. Such an innovation is so vast, however, that its endorsement or otherwise requires deliberations that lie far beyond our terms of reference. What these terms do enable us to address, however, is the question of what industrial diseases are most appropriately and equitably encompassed by a framework of workers' compensation as distinct from one of general compensation. The broader coverage inherent in a general compensation scheme occasions higher costs and hence exerts a downward push on the level of benefits. This means that when consideration is given, in the realm of disease, to substituting general compensation for workers' compensation, the rationale must be that the universality of lower compensation, whatever the cause of the disease, is preferable to the higher compensation reserved for the select few whose disease was caused by the workplace. On the other hand, when consideration is given to retaining workers' compensation for victims of industrial disease, the rationale must be that the victims of industrial disease are entitled in equity to the same level of compensation as the victims of industrial accidents.

The weight we accord to the latter consideration is already evident from our recommendation that asbestosis and mesothelioma should be listed in Schedule 3. These are instances where we are satisfied that the relationship between disease and occupational exposure is substantially clear. The case of asbestos-related cancers is different. It may be that, when all the stipulations incorporated in the current guidelines are met, there are grounds for a strong presumption that the cancer was asbestos-induced. However, the issue of lifestyle remains clearly present, given in particular the much higher incidence of lung cancer among asbestos workers who are smokers.

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[65] Weiler, *Protecting the Worker from Disability: Challenges for the Eighties*, chap. 3.

Unless it were to subsume workers' compensation altogether, a general compensation scheme will not obviate the need for eligibility rules that will seek to delineate those diseases that should in equity be subject to workers' compensation. Where exposure to a substance in an industrial process is necessary and sufficient for the employee to contract the disease, the eligibility rules should be incorporated into the workers' compensation statute through Schedule 3. Where the grounds are less compelling, eligibility rules more similar to the current Board guidelines would be suitable. The large number of remaining instances could properly be left to the general compensation scheme. The setting of the eligibility rules places a premium on judgement.

The matter of judgement, of course, is immensely more consequential and delicate in the absence of a general compensation scheme. In prescribing a process for devising eligibility rules, we do not consider ourselves entitled to assume that such a major social policy innovation will soon be in place. And even if general compensation became a reality, a formal, systematic, and comprehensive mechanism to design and review the eligibility rules that structure the Board's discretion would remain imperative so long as a separate category of workers' compensation remained in effect.

As we view them, eligibility rules, by structuring the Board's discretion in the matter of industrial disease compensation, serve three functions. The first is to promote certainty and simplicity in claims adjudication by declaring that any claims that fall within their terms will be compensated, subject only to a verification of the facts of each individual case. The second is to promote horizontal equity (the equal treatment of equals) by ensuring that similarly situated individuals, namely, all claimants who fall within the rules, will be treated similarly. The third is to serve as building-blocks of public policy with respect to industrial disease in that eligibility rules are a declaration that a specific disease under stipulated circumstances will be regarded as compensable.

We expressly wish to underline the importance we attach to these three functions by using the more formal term "eligibility rules" to encompass what the Board currently calls "guidelines." Especially because eligibility rules involve equity among citizens and constitute declarations of public policy, we urge that they should be determined by an open process. To be sure, openness may also enhance the technical quality of the rules in that a wider array of expert views may be brought to bear on their content. The contrary view is that openness will dilute the quality of the guidelines because they will be the outcome of conciliation between conflicting vested interests. Whatever the merits of these contending views, considerations of equity and public policy speak compellingly in favour of an open process.

To formalize and open the process of devising and reviewing eligibility rules, we advocate the creation, by statutory amendment to the *Workers' Compensation Act*, of an Advisory Council on Industrial Disease Policy (ACIDP). Our choice of name is explicit and deliberate for two reasons. First, the name is meant to emphasize that eligibility rules are indeed matters of public policy. Second, it is intended to convey that any of a number of industrial disease matters should properly come within the Council's purview. We shall develop examples of such matters in Chapter 14.

The Advisory Council on Industrial Disease Policy should have an arm's length relationship to the Workers' Compensation Board. We make the gratifying observation that the Board's Vice-Chairman of Administration explicitly endorsed the concept of such a body in his testimony before us.[66] The ACIDP should be composed of a Chairman and not more than eight part-time members appointed by the Lieutenant Governor in Council. The members should be representative of the views of labour, management, the public, and the medical-scientific community. We specify this breadth of membership because the formulation of advice on industrial disease policy is as much a matter of pragmatic judgement as of scientific judgement.

In the matter of eligibility rules, so as to ensure that the most relevant scientific judgement will be brought to bear, the ACIDP should be empowered to appoint disease-specific "Panels" composed of a small number (normally two or three) of medical-scientific experts. Once constituted, a Panel would cause a study or studies to be undertaken by recognized specialists, here following a step the Board has taken in developing its guidelines. Upon receiving the study, the Panel would draft suggestions for an eligibility rule and convey these to the ACIDP along with the study. If the Panel could not find grounds for an eligibility rule, it would so inform the ACIDP, stating the reasons in its report.

We advocate that written comments on the Panel's report should be solicited by the ACIDP. The ACIDP would consider these comments but, so that the rule-setting process not be overly time-consuming, it should not be obligated to hold hearings. The ACIDP would then submit its formal advice to the Corporate Board and include in its advice either a specific eligibility rule or the reasons why such a rule should not be promulgated. Both the advice and the Board's response should be public, the advice being published at the same time as the response. The Board should be able to accept, reject, or ask for reconsideration. In the latter two eventualities, the Board's published response should state reasons.

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[66] RCA Transcript, Evidence of Mr. Alan G. MacDonald, 11 August 1982, Volume no. 55, p. 16.

While we view an arm's length relationship between the ACIDP and the Corporate Board as essential, we consider open linkages between the two to be equally necessary. The importance of these linkages rests on the Board's responsibility for claims adjudication. It is the case-by-case determination of claims that appear unusual or historically new which can often unearth the initial patterns of information that in time suggest the possibility of structuring discretion through an eligibility rule. For this reason, the legislation should specify that the Board should be able to request the ACIDP to initiate the contemplation of an eligibility rule. Furthermore, eligibility rules should be drafted in a manner that is sensitive to the realities of the adjudication process; they should be understandable and understood by both claimants and adjudicators. Again, in that these rules are building-blocks of industrial disease policy, they should not be divorced from first-hand knowledge of the overall policies and practices of the Board. For these reasons, we advocate that the Chairman of the ACIDP be an *ex officio* Director of the Corporate Board.

In light of all the above considerations, we recommend that:

12.11 The Workers' Compensation Act should be amended to provide for the creation of an Advisory Council on Industrial Disease Policy responsible for advising the Corporate Board on eligibility rules regarding disease compensation and on other matters of industrial disease policy. The Council's advice should be developed either as a matter of its own initiative or in response to an explicit request from the Corporate Board. The Council should be appointed by the Lieutenant Governor in Council and be composed of a Chairman, who would be an ex officio Director of the Corporate Board, and not more than eight members appointed for stated terms. The legislation should authorize the Council to appoint expert Panels from time to time and should stipulate that both the Council's advice and the Corporate Board's response be public documents.

With respect to the Board's current guidelines on asbestos-related cancers, we explicitly observed earlier in this chapter that whatever the specific merits of their content, they have served to structure the Board's discretion. Furthermore, the Board's benefit of doubt policy appears to have been applied by adjudicators to claims outside the guidelines in a manner that does not reveal a negative predisposition. We have no doubt, however, that the current guidelines will be improved when they are transformed into eligibility rules through the process we have prescribed. By no means do we confine this observation to the medical content of the guidelines. Thus, for example, all three guidelines refer to the need to establish a "clear and adequate" history of exposure to asbestos dust. In both his study and his testimony, Professor Barth openly wondered about the meaning of the words "clear and adequate."[67] The Executive Director of the Board's Medical Services Division, in his written response to the Barth study, defended these words as superior to such terms as "significant," "definite," "definitive," or "sufficient."[68] Whatever the merits of this response, we view both the choice and meaning of all such terms as matters that demand precisely the perspectives of pragmatic judgement that the Advisory Council on Industrial Disease Policy can bring to the setting of eligibility rules through the open process we have prescribed. Accordingly, we recommend that:

12.12 The Advisory Council on Industrial Disease Policy should, as a matter of high priority, transform the current Board guidelines on asbestos-related cancers into eligibility rules in accordance with the process prescribed in this Report.

C.4 Breaking New Ground: The Eligibility of Family Members for Workers' Compensation

In Chapter 5 we assess the evidence concerning the possibility that the family members of an asbestos worker can contract asbestos-related disease from the dust that the worker brings into his domicile. The world health literature reveals no known cases of asbestosis among family members. This is consistent with the conclusion that asbestosis does not develop at low levels of occupational exposure. There may be instances of cancer that could arouse suspicion of family exposure to asbestos, but the suspicion will be in the realm of hypothetical possibility. This is consistent with the extent to which occupational exposure to asbestos, especially at lower levels, leaves the issue of the work-relatedness of cancer entangled with other sources of causation. This leaves mesothelioma. Unhappily, as Chapter 5 makes clear, the incidence of this disease among family members of asbestos workers is clearly recorded in the health literature.

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[67] Barth, *Workers' Compensation and Asbestos in Ontario,* pp. 5.12, 5.16; and RCA Transcript, Evidence of Professor Peter S. Barth, 24 August 1982, Volume no. 57, p. 81.

[68] McCracken, "Comments and Critique on a Study Prepared by Peter S. Barth, *Workers' Compensation and Asbestos in Ontario,*" p. 6. See also, letter from Peter S. Barth, Professor of Economics, University of Connecticut to the Royal Commission on Asbestos, 2 September 1982.

Neither our research nor our testimony has unearthed a case of mesothelioma in Ontario that could be clearly attributed to family exposure. Disturbingly, however, the Board's Chest Disease Specialist, Dr. Douglas W. Dyer, shared with us during testimony his suspicion that a mesothelioma claim he had recently examined might in fact be a case of family exposure. The claimant had worked for the same company as his father, who himself had worked for this company for a number of years. But the claimant's (i.e., the son's) latency period ". . . was more in keeping with a family exposure than his actual work exposure."[69] In this instance, the claim was granted in line with the Board's practice of compensating mesothelioma victims whenever it has evidence of occupational exposure to asbestos.

Should Ontario break new ground by extending workers' compensation to the members of workers' families who contract disease from a substance to which the worker has been occupationally exposed? To our knowledge, the asbestos-related compensation bills before the United States Congress do not currently envisage coverage for family members of asbestos workers. The Johns-Manville Corporation has lobbied the Congress to extend coverage to family members for the apparent purpose of limiting its tort liability.[70] If nothing else, this is a potent reminder that workers' compensation serves the interests of business fully as well as the interests of labour.

The fundamental point on which we base our own considerations is the workplace-relatedness of a disease. We have declared ourselves satisfied that the evidence linking occupational exposure to asbestos with mesothelioma is sufficiently robust to warrant the inclusion of mesothelioma in Schedule 3. Our assessment of the medical evidence in Chapter 5 is such that, at least on the basis of balance of probabilities, we would associate a mesothelioma diagnosed in a family member who had shared the domicile of a worker while that worker was occupationally exposed to asbestos with such occupational exposure. In equity, we conclude that workers' compensation should be extended, in mesothelioma cases, to family members who have been domiciled with an asbestos-exposed worker at the time of the worker's exposure. This conclusion is in line with other opinions, notably that of Professor Barth.[71]

Having reached this conclusion, we address the practical question of the level of compensation for which family members should be eligible. In all instances, given the workplace relatedness of mesothelioma, equity demands that the level of compensation flowing from the death of a family member from this disease should be the same as the compensation that flows under the Act from the death of an employee. The present Act does not relate survivor benefits to employee income, but such a relationship is envisaged in the *White Paper*. Should such a relationship be enacted, the income of the deceased family member should be deemed the employee's income for the purpose of calculating survivor benefits.

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[69] RCA Transcript, Dr. Douglas W. Dyer, during evidence of Mr. John F. McDonald, 14 July 1982, Volume no. 49, p. 154.

[70] RCA Transcript, Evidence of Professor Peter S. Barth, 24 August 1982, Volume no. 57, pp. 94–95.

[71] Ibid., p. 94. See also, Peter S. Barth with H. Allan Hunt, *Workers' Compensation and Work-Related Illnesses and Diseases* (Cambridge, Mass.: The MIT Press, 1980).

As for the question of financing the benefits awarded, we are loath to envisage a situation where the cost of family member claims must invariably be borne by the Board's accident fund and apportioned among employers generally. The contracting of an industrial disease by a family member, if it ever occurs, will be a rare and poignantly tragic occasion; even though the resulting costs will be trivial in relation to global compensation costs, such an instance should be highlighted because of its exceptional nature. It follows that the Board should acquire, by amendment to the *Workers' Compensation Act*, a statutory right to recover the costs of any benefits paid with respect to family members from the individual employer involved.

In line with the above considerations, we recommend that:

12.13 The Workers' Compensation Act should be amended so as to:

(i) entitle individuals who contract mesothelioma, and are family members of asbestos workers who were domiciled with these workers at the time such workers were occupationally exposed to asbestos, to the same compensation benefits as the Act accords to employees; and
(ii) vest in the Workers' Compensation Board a statutory right to recover the costs of benefits paid to a family member of an asbestos worker from the employer of that worker.

Appendix to Chapter 12
Ontario Workers' Compensation Board Guidelines for Adjudication

Lung Cancer in Asbestos Workers: Guidelines for Adjudication

1. That lung cancer in asbestos workers be accepted as an industrial disease under Section 118 [now Section 122] and Section 1(1)(l) [now Section 1(l)(n)] of the Act as peculiar to and characteristic of a process, trade, or occupation involving exposure to asbestos.

2 . That based on medical studies, lung cancer claims be favourably considered when the following circumstances apply:

2.1 There is a clear and adequate history of at least 10 years' occupational exposure to asbestos.

2.2 There is a minimum interval of 10 years between first exposure to asbestos and the appearance of lung cancer.

2.3 Claims which do not meet the guidelines in 2.1 and 2.2 should be individually judged on their own merit having regard to the intensity of exposure and other factors peculiar to the individual case. The benefit of reasonable doubt applies.

NOTE: These guidelines do not apply to mesothelioma claims.

Approved by the Board April 13, 1976.

Mesothelioma in Asbestos Workers: Guidelines for Adjudication

1. That mesothelioma in asbestos workers be accepted under Section 118 [now Section 122] and Section 1(1)(l) [now Section 1(1)(n)] of the Act as peculiar to and characteristic of a process, trade, or occupation involving exposure to asbestos.

That based on medical studies, mesothelioma claims be favourably considered when the following circumstances apply:

1.1 There is a clear and adequate history of at least 10 years' occupational exposure to asbestos.

and —

1.2 There is a minimum interval of 15 years between first exposure to asbestos and the appearance of mesothelioma.

1.3 Claims which do not meet the guidelines in 1.1 and 1.2 should be individually judged on their own merit having regard to the intensity of exposure and other factors peculiar to the individual case. Consideration will be given where it seems evident that the mesothelioma cancer resulted from occupational exposure to asbestos. The benefit of reasonable doubt applies.

Approved by the Board April 13, 1976.

Gastrointestinal Cancer — Asbestos Workers: Guidelines for Adjudication

1. That gastrointestinal cancer in asbestos workers be accepted as an industrial disease under Section 118 [now Section 122] and Section 1(1)(l) [now Section 1(1)(n)] of the Act as peculiar to and characteristic of a process, trade, or occupation involving exposure to asbestos.

2. That based on medical studies, gastrointestinal cancer (esophagus, stomach, small bowel, colon, and rectum) be favourably considered when the following circumstances apply:

2.1 There is a clear and adequate history of occupational exposure to asbestos dust, and while such occupational exposure cannot be quantitatively described, it should be of a continuous and repetitive nature and should represent or be a manifestation of the major component of the occupational activity.

2.2 There is a minimal interval of 20 years between first exposure to asbestos and the diagnosis of the gastrointestinal cancer.

2.3 That all primary cancers associated with the esophagus, stomach, small bowel, colon, and rectum be included in the classification of gastrointestinal cancers.

2.4 That no distinction be given as to the site of the cancer in assessing the merit of a claim.

2.5 Claims which do not meet the guidelines in 2.1, 2.2, 2.3, 2.4 should be individually judged on their own merit having regard to the nature of the occupation, the extent of the exposure, and other factors peculiar to the individual case. Consideration will be given where it seems evident that the gastrointestinal cancer resulted from occupational exposure to asbestos. The benefit of reasonable doubt applies.

Approved by the Board October 7, 1976.

Guidelines for Adjudication of Claims for Laryngeal Cancer in Industry Related to Asbestos Exposure and Nickel Exposure

Recommendation

1. Laryngeal cancer in workers occupationally exposed to asbestos fibre and/or to nickel aerosol in specific industrial processes be accepted as an industrial disease under Section 118 [now Section 122] and Section 1(1)(l) [now Section 1(1)(n)] of the Act as peculiar to and characteristic of such processes.

2. Based on medical studies, claims for laryngeal cancer be favourably considered under the following circumstances:

2.1 Any industrial process in the nickel industry which produces nickel in aerosol dispersion whether in combined or elemental form. This may include the following:
— Roasting
— Smelting
— Refining
— Welding
— Electroplating.

2.2 Any occupation in which there is a clear and adequate history of occupational exposure to asbestos dust, and while such occupational exposure cannot be quantitatively described, it should be of a continuous and repetitive nature and should represent or be a manifestation of the major component of the occupational activity.

3. Duration of Exposure

3.1 *Nickel* — an accumulative minimum of 15 years' exposure to nickel aerosols as defined in 2.1.

3.2 *Asbestos* — an accumulative minimum of 10 years' proven exposure as defined under 2.2.

3.3 *Nickel and Asbestos* — an accumulative minimum of 7.5 years' nickel as well as 5 years' asbestos exposures in the case of dual exposure.

4. Inception Period

4.1 *Nickel* — this shall be a minimum of 20 years from the commencement of the first hazardous exposure.

4.2 *Asbestos* — this shall be a minimum of 20 years from the commencement of the first hazardous exposure.

4.3 *Nickel and Asbestos* — this shall be a minimum of 15 years from the commencement of the first hazardous exposure.

5. Claims which do not meet the guidelines should be individually judged on their own merit having regard to the intensity of exposure and other factors peculiar to the individual case. The benefit of reasonable doubt applies.

Approved by the Board May 4, 1978.

SOURCE: The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, pp. 23, 25, 30, 43.

Chapter 13 Processing Asbestos-Related Claims: Procedural and Substantive Issues

A. The Claims Process: An Overview

Asbestos-related claims that reach the Workers' Compensation Board originate from a variety of sources. Of the claims surveyed by Professor Peter S. Barth in his study for this Commission, 41% originated from employees or their survivors, 31% from unions, 10% from employers, and 18% from physicians.[1] The framework within which the Board handles asbestos-related claims differs depending on whether the claim involves: (i) cancer or mesothelioma; (ii) asbestosis; or (iii) the death of an individual who was in receipt of a permanent disability pension for asbestosis at the time of death. We shall begin by considering the framework for processing cancer and mesothelioma claims. This is the simplest of the three processing frameworks. It involves the guidelines and the benefit of doubt policy described in Chapter 12. Also, it shares important elements in common with the other two claim categories and hence provides an introductory overview of the process followed by all asbestos-related claims.

In common with the other categories, cancer and mesothelioma claims pass initially into the Claims Adjudication Branch of the Board's Claims Services Division. There, a claims adjudicator in the Industrial Diseases and Dependants Section proceeds to build a file by assembling information from the claimant, the claimant's physician, and the employer. Prescribed forms are available for all three parties. Particularly in cancer cases, the adjudicator may file a physician's letter or report instead of a form.[2] For reasons upon which Professor Barth elaborates, the employer form may be a source of delay if the claimant has indicated asbestos exposure in more than one employment, or if the employer no longer exists or has inadequate records from which to establish exposure.[3] In such cases, the Claims Adjudication Branch can call upon the resources of the Investigations Services Section of the Claims Administrative Services Branch.

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[1] Peter S. Barth, *Workers' Compensation and Asbestos in Ontario*, Royal Commission on Asbestos Study Series, no. 2 (Toronto: Royal Commission on Asbestos, 1982), pp. 2.1–2.2.

Once compiled, the claim file moves from the Claims Adjudication Branch to the Medical Branch of the Medical Services Division. There it passes into the hands of one of two Board physicians: Dr. Charles Stewart, whose title is Chest Disease Consultant, or Dr. Douglas W. Dyer, whose title is Chest Disease Specialist. The Board physician assesses the evidence regarding asbestos exposure and may request additional information from the claims adjudicator or the Investigations Services Section. He may himself, as did Dr. Dyer in the celebrated case of the war-time Ottawa gas mask workers, undertake extraordinary efforts to identify both the source of asbestos exposure and other potential claimants.[4] The other function of the Board physician is to assess whether the claimant's diagnosis has been correctly established by reviewing the opinion of the claimant's physician, hospital records, and tissue records. In a number of cases, the Board physician may refer pathologists' slides or uncut tissue to Dr. Alexander C. Ritchie, Professor of Pathology at the University of Toronto, for an expert opinion. Finally, the Board physician determines whether or not the diagnosis and the exposure indicate that the claim is one of asbestos-caused cancer or mesothelioma and so informs the Claims Adjudication Branch.

If the finding of the Board physician is positive, the claims adjudicator allows the claim. Given the terminal nature of cancer and mesothelioma, the claims that are allowed almost invariably involve survivor benefits. According to Professor Barth, about one in six of the cancer claims allowed have involved some temporary total or permanent disability benefits as well as survivor benefits.[5] The claims adjudicator calculates the amount of the disability or survivor benefits payable under the provisions of the *Workers' Compensation Act*, and makes the award.

If the Board physician has made a negative finding, the claims adjudicator reports to his team co-ordinator within the Industrial Diseases and Dependants Section of the Claims Adjudication Branch.[6] The team co-ordinator may overrule the adjudicator or direct that further inquiry take place, but Professor Barth found only one instance in which a negative finding that involved an asbestos-related disease was challenged at the level of the Claims Adjudication Branch.[7] However, this Branch, under Board policy, does not have the authority to deny a claim on the basis of a negative finding. The claim must therefore be sent to the Claims Review Branch before it can be officially denied. Professor Barth found virtually no reversals of negative findings at the level of the Claims Review Branch, and the Board's Chest Disease Consultant could recall no instance in which this had occurred.[8] Upon confirmation by the Claims Review Branch, the claimant (or the claimant's survivor) is notified that the claim has been denied. At this point, the primary adjudication phase of the claims process has ended.

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[2] Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, pp. 68–69.

[3] Barth, *Workers' Compensation and Asbestos in Ontario*, p. 2.4.

[4] RCA Transcript, Evidence of Dr. Douglas W. Dyer, 16 July 1982. Volume no. 51, pp. 108–119.

[5] Barth, *Workers' Compensation and Asbestos in Ontario*, p. 6.42.

[6] RCA Transcript, Evidence of Mr. John F. McDonald, 13 July 1982, Volume no. 48, p. 42.

An unsuccessful claim that is appealed will be reviewed by an appeals adjudicator under the Board's Registrar of Appeals. This is the first echelon of the Board's appeal structure. Whether or not the appeals adjudicator holds a hearing, he refers the evidence before him back to the Medical Services Division. We note that in his testimony the Board's Chest Disease Consultant, Dr. Charles Stewart, could not bring to mind a cancer claim concerning which he had varied his original opinion as a result of the evidence before the appeals adjudicator.[9] Furthermore, he could recall only one occasion on which his initial recommendation had been reversed by an appeals adjudicator, and in this instance the claim was not one involving asbestos-related cancer.[10]

We have already recommended, in Chapter 12, that the appeals structure above the first echelon of appeals should be completely overhauled. Therefore, our descriptive overview can appropriately terminate at the level of the appeals adjudicator.

What stands out from this overview is the dominant role, duly noted by Professor Barth, of the Board's physicians. In large part, we view this as flowing naturally from the simple fact that diagnosing cancer, mesothelioma, or any disease is a matter of medical knowledge. Establishing exposure to asbestos, for its part, is not intrinsically a medical matter. We acknowledge, however, that medical knowledge is of enormous assistance in understanding the literature which associates disease with hazardous workplace substances. It is this literature in turn that has infused the development of the Board's cancer and mesothelioma guidelines, with whose terms the Board physicians are intimately acquainted. Our judgement is to accept as realistic the likelihood that these physicians will dominate the primary adjudication of mesothelioma and cancer claims and to attempt to structure this dominant role rather than undermine it.

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[7] Barth, *Workers' Compensation and Asbestos in Ontario*, p. 2.6.

[8] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, p. 71.

[9] Ibid., p. 72.

[10] Ibid., pp. 126–127.

The degree of structuring that we deem desirable is basically encompassed by the changes we have prescribed in Chapter 12, notably: (i) making mesothelioma a scheduled disease; (ii) bringing eligibility rules for cancer under the purview of an Advisory Council on Industrial Disease Policy; (iii) giving claimants statutory access to any and all medical records; and (iv) replacing the current upper echelon of the Board's appeal structure with a Workers' Compensation Appeals Tribunal that incorporates Medical Review Panels.

Our descriptive overview of the claims process gives us further reason to emphasize the importance of Medical Review Panels. We look on these repositories of outside medical knowledge as the necessary means of countervailing the enormous internal influence of Board physicians in the primary adjudication process. Also, we permit ourselves to expect that as written appeals decisions, coupled with Medical Review Panels, grow in volume, the Claims Review Branch and the first-echelon appeals adjudicators will assimilate information that should make them more knowledgeable reviewers of the findings of Board physicians.

Even under our recommendations, it is our view that claims for cancer, other than mesothelioma, will remain the most problematic area of asbestos-related disease compensation. However constructively eligibility rules may be refined, there will be a need to ensure that Board officials adjudicate claims that lie outside the rules with an open mind. We cite in Chapter 12 quantitative evidence which indicates that Board physicians have not approached asbestos-related lung cancer claims with a negative mind-set. We take this as indicating that the Board's benefit of doubt policy has influenced its physicians. Whether this policy is being consistently followed is another matter. While we have not ourselves reviewed the details of individual claims, we acknowledge that the impartial reassessment of individual cancer cases conducted by Dr. Annalee Yassi, for Professor Paul C. Weiler's inquiry into workers' compensation in Ontario, reveals outcomes whose consistency she questions.[11] The problem may lie in the fact that the content of the benefit of doubt policy is not explicitly spelled out. We shall address this question at the end of this chapter. Presently, let us turn to a claims category that raises peculiar issues of procedure and substance: asbestosis.

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[11] Annalee Yassi, "Occupational Disease and Workers' Compensation in Ontario: A Report for the Weiler Inquiry," Toronto, Ontario Ministry of Labour, in press.

B. Processing Asbestosis Claims

B.1 Introduction

Asbestosis is an asbestos-specific form of pneumoconiosis that is irreversible and normally progressive. Asbestosis can be a direct cause of death; more generally, its victims are susceptible to death from related causes and have a decreased life expectancy. Dr. Murray M. Finkelstein of the Ministry of Labour, the principal author of a study of Ontario workers who have been compensated for asbestosis, testified before us that the survival rate of compensated asbestotics was only 70% of that expected in Ontario males of identical age five years after compensation and 53%, ten years after compensation.[12]

Its implications for life expectancy aside, asbestosis is a chronic and disabling condition. This means that in the realm of compensation, asbestosis constitutes a classic instance of a workplace disease whose sufferers are eligible for *partial* disability benefits. It follows that the compensation of asbestosis requires an important additional judgement: besides exposure and diagnosis, the quantum (percentage) of impairment must be determined.

All of the officials involved in the processing of cancer and mesothelioma claims reappear in the processing of asbestosis claims, namely, the adjudicators of the Claims Adjudication Branch, the Board physicians of the Medical Services Division, the personnel of the Claims Review Branch, and the appeals adjudicators. However, two additional participants play important roles: the Chest Surveillance Programme of the Occupational Health Branch of the Ministry of Labour and the Board's Advisory Committee on Occupational Chest Diseases (ACOCD). These roles overlap. Furthermore, the role of the ACOCD is an optional one in the sense that the physicians of the Medical Services Division may choose not to send a claim to the ACOCD. Let us begin by situating all this in the context of the claims process.

B.2 The Chest Surveillance Programme, the Medical Services Division, and the Advisory Committee on Occupational Chest Diseases

An asbestosis claim, like other disease claims, reaches the Board's Medical Services Division once a file has been compiled by the Claims

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[12] RCA Transcript, Evidence of Dr. Murray M. Finkelstein, 28 July 1981, Volume no. 24, p. 12. See also, Murray M. Finkelstein, Robert Kusiak, and George Suranyi, "Mortality Among Workers Receiving Compensation for Asbestosis in Ontario," *Canadian Medical Association Journal* 125 (1 August 1981): 259-262.

Adjudication Branch. The processing of such a claim is subject to what the Board variously calls "procedural guidelines"[13] or "guidelines for adjudication."[14] In effect, these call for: (i) a "clear and adequate history" of occupational exposure to asbestos; (ii) a diagnosis of "frank asbestosis"; and (iii) the establishment of "the degree of functional impairment" by the ACOCD.

The Board physician begins by addressing the evidence regarding asbestos exposure and the nature of the diagnosis. At this point, the Chest Surveillance Programme enters the scene.

Originally through the Ministry of Health, latterly through the Ministry of Labour, this Programme has surveyed asbestos workers since 1947 on regular cycles. The Programme routinely forwards individual reports on each worker to the plant physician.[15] Chest Surveillance reports will therefore commonly accompany the physician's form that is in the asbestosis claimant's file. If they do not, the Board physician may search for relevant copies of the reports. These copies are filed in the Medical Services Branch by company or industry,[16] but are not available to the Claims Adjudication Branch.[17]

Whether or not he has Chest Surveillance reports, the material the Board physician is seeking to assess is x-ray evidence and evidence of pulmonary function deficiency. In the absence of Chest Surveillance reports, or by way of supplementing them, such evidence may have come from the claimant's physician or from a hospital radiologist or respirologist.[18] On occasion the Board physician will himself procure x-ray reports.[19]

At this juncture the Board physician exercises a choice. He may forward the claim to the ACOCD, or he may return it to the claims adjudicator with a negative finding. Because the Chest Surveillance Programme encompasses all employers known by the Ministry of Labour to be engaged in industrial processes that involve the use of asbestos, its reports are most helpful in establishing the fact that the worker has been exposed to this substance. Furthermore, the Programme's x-ray evidence and pulmonary function tests lend "great strength"[20] to the Board physician's assessment of a possible diagnosis of asbestosis. As likely as not, Chest Surveillance reports may be associated with a negative finding by the Board physician. After sampling 40 claims that had been returned to the claims adjudicator with a negative finding, the Board's Chest Disease Consultant testified before us that 27 included Chest Surveillance reports.[21]

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[13] The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, p. 6.

[14] Ontario, Royal Commission on Asbestos, Exhibit IV-1 [hereafter RCA Exhibit], in RCA Transcript, Evidence of Mr. John F. McDonald, 13 July 1982, Volume no. 48: The Workmen's Compensation Board, Ontario, Assorted Policy Statements and Forms.

[15] Ontario, Ministry of Labour, Written submission to the Royal Commission on Asbestos, #43, February 1981, p. 59.

[16] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, p. 146.

[17] Ibid., p. 126.

[18] Ibid., pp. 12-13.

[19] Ibid., p. 25.

[20] Ibid., p. 13.

This testimony offers a counterpoint to Professor Barth's observation that a claim is less likely to evoke a positive response from Board physicians if the only medical evidence involved has come from the claimant's physician.[22] More importantly, in our view, it highlights the Board physician's role as the gatekeeper to the ACOCD. Whatever the source of the medical evidence in the file, it is clear that the Board physician possesses enormous discretion with respect to the disposal of an asbestosis claim. Professor Barth's study supplied data covering 391 asbestosis claims initiated from 1970 through 1980; 109 of these claims were the subject of negative findings by Board physicians and not forwarded to the ACOCD.[23]

To our knowledge, a negative finding by the Board physician is invariably accepted by the claims adjudicator. Unlike the situation that prevails in cancer claims, however, the Claims Review Branch may question the Board physician's negative finding. Of course, what is involved is not whether to deny or grant the claim, but whether to deny the claim or forward it to the ACOCD. The Board's Chest Disease Consultant testified that the Claims Review Branch, in questioning a negative recommendation, ". . . may have some comment on the duration of exposure, or they may point out that the family doctor has strong views or something."[24] If his negative recommendation is questioned, the Board physician will ". . . not put up any fight . . . if there was insistence,"[25] and he will forward the questioned claim to the ACOCD. Let us examine the ACOCD.

The ACOCD acquired its name in 1968, but at that time already had a prior existence of forty years' duration as the Silicosis Referee Board. By 1968, the Workers' Compensation Board had long made it a practice to send to the Silicosis Referee Board claims involving chest diseases other than silicosis, and the change of name was executed to reflect this fact and also to confirm that the work was more specifically advisory than similar to that of a medical referee as defined in the *Workers' Compensation Act*.[26] In addition to asbestosis, the ACOCD deals with any claims that may relate to mineral dust disease, for example, silicosis, talcosis, hard-metal disease, nepheline syenite, pneumoconiosis, Shaver's Disease, and bauxite pneumoconiosis.[27] It also deals with certain trauma cases involving pulmonary injury.[28] According to the Board's Chest Disease Consultant, the ACOCD processes 420 to 450 claims per year.[29] On the basis of Professor Barth's data, 5 to 10% of these claims would be for asbestosis.[30]

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[21] Ibid., p. 8.

[22] Barth, *Workers' Compensation and Asbestos in Ontario*, pp. 2.14–2.15.

[23] Ibid., p. 6.27.

[24] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, p. 31.

[25] Ibid.

[26] R.S.O. 1980, c. 539. Medical referees are covered by ss. 1(1)(s) and 22 of the Act; the current statutory basis of the ACOCD is found in s. 71(3)(g). See RCA Transcript, Evidence of Mr. John F. McDonald, 13 July 1982, Volume no. 48, p. 59.

The membership of the ACOCD is appointed by the Executive Director of the Board's Medical Services Division for indefinite terms of office.[31] It numbers seven individuals, although there is confusion regarding who is a member and who is a consultant.[32] Of the physicians who were members of the ACOCD during the 1970s, three were also active in the Chest Surveillance Programme. The significance of this overlap will become apparent as our narrative proceeds.

Upon receiving the claims file from the Board physician, the ACOCD has two tasks. The first is to confirm or deny a diagnosis of asbestosis. The second, which accompanies an affirmative diagnosis, is to establish the quantum (percentage) of the claimant's impairment. Of course, the Board physician, in his gatekeeper role, has already made a negative diagnosis for the asbestosis claims he has not forwarded to the ACOCD. The ACOCD's first task is therefore pertinent only to a subset of the asbestosis claims that have reached the Board (according to Professor Barth's data, 282 of the 391 claims initiated from 1970 to 1980).[33]

The ACOCD's standard procedure begins with a clinical examination of the claimant, conducted by one of the Committee's members. On any initial claim, the examination is always held at the Medical Service Chest Clinic maintained by the Occupational Health Branch of the Ministry of Labour at 880 Bay Street in Toronto. (This Clinic was formerly called the Chest Diseases Clinic and was located at 50 Grosvenor Street in Toronto.)[34]

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[27] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, p. 32.

[28] Ibid., p. 43.

[29] Ibid.

[30] Barth, *Workers' Compensation and Asbestos in Ontario,* p. 6.27.

[31] The indefinite term of office can be tantamount to a lifetime appointment; one physician held office from 1929 to 1973, when he retired. See RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, pp. 41–42.

[32] The Board's Chest Disease Consultant considers the five physicians on the ACOCD who regularly examine patients and two others who join in its meetings to be members of the Committee. See RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, p. 37. One of the physicians in the latter category, Dr. Cameron C. Gray, considers himself a consultant rather than a member because he does not examine claimants but only meets with the Committee. The other physician who is sometimes referred to as a consultant, Dr. David C.F. Muir, occasionally examines claimants. See RCA Transcript, Evidence of Dr. Cameron C. Gray, 16 July 1982, Volume no. 51, pp. 4–5.

[33] Barth, *Workers' Compensation and Asbestos in Ontario,* p. 6.27.

[34] RCA Transcript, Evidence of Dr. Charles Stewart, 15 June 1982, Volume no. 50, p. 44.

It will be recalled that the Occupational Health Branch is responsible for the Chest Surveillance Programme.

The clinical examination takes about three hours.[35] The taking of the claimant's history may add to the exposure information that is already on file.[36] The claimant is physically examined and x-rayed. Pulmonary function tests and exercise tests are given. If the examining physician deems it necessary, he may refer the claimant to a hospital for a lung biopsy.[37] Testifying on the basis of some ten years' experience as consultant to the ACOCD, Dr. Cameron C. Gray told us: ". . . I have always been impressed with the detail that has been taken and recorded by the examining physician with respect to the symptoms, emphasizing those symptoms that have to do, of course, with the chest — shortness of breath being the one that's probably the most stressed — but also cough sputum, pain, wheezing, so on."[38]

The members (and consultants) of the ACOCD meet weekly as a committee to review the claims before them. Each claim is presented by the physician member who conducted the claimant's physical examination. To summarize from Dr. Gray's testimony,[39] the occupational background of the claimant is presented, and then the person's radiographs are put up on a screen. The ACOCD commonly has the advantage of a sequential series of x-rays taken from the time the individual started work; the source of this sequential series will normally be the Chest Surveillance reports. Then, in Dr. Gray's words, ". . . the pulmonary function studies are reported and discussed, the electrocardiogram has usually been taken in these individuals and that, too, is presented and discussed; and with those major bits of evidence of history — the radiograph, the pulmonary function studies, and cardiogram — it is then that our deliberations take [place] as to, 'Does the man have the diagnosis? If so, does he have any impairment of function because of the diagnosis?' "[40]

The presenting physician who examined the claimant may terminate his presentation by expressing an opinion concerning the presence or absence of impairment, but this opinion is only by way of initiating the Committee's discussion.[41] The Committee comes to a single decision, normally by consensus. The occasional vote may be taken, but neither the vote nor the disagreement is recorded. In the event of a very close vote, the Committee may decide to postpone its decision and re-examine in six months.[42] Disagreement, when it occurs, most likely involves claims in which the quantum of impairment, if any, is thought to be low.[43] If asbestosis has been diagnosed, the quantum of impairment is determined on a scale of from 10 to 100%, normally in increments of 10 percentage points, but occasionally more closely refined to, for example, 35% or 75%.[44]

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[35] Ibid., pp. 43-44.

[36] Ibid., p. 46.

[37] RCA Transcript, Evidence of Dr. Jerome J. Vingilis, 8 June 1982, Volume no. 39, p. 42.

[38] RCA Transcript, Evidence of Dr. Cameron C. Gray, 16 July 1982, Volume no. 51, p. 11.

[39] Ibid., pp. 11-12.

[40] Ibid., p. 12.

[41] Ibid.

The official finding of the ACOCD, either to deny the claim because the diagnosis of asbestosis is negative, or to allow the claim at a specified quantum of impairment, is recorded and signed by the physician who examined the claimant.[45] It is forwarded to the Board physician, citing reasons for the finding, over the letterhead of the Ministry of Labour at whose clinical examination facilities the claimant was examined and the ACOCD convened.[46] The Board's Chest Disease Consultant may occasionally reply to the ACOCD with a request for clarification, but could not recall in testimony any occasion on which he could not accept a Committee report.[47] He returns the claim file, including the ACOCD report, to the Claims Adjudication Branch. There the adjudicator, if the ACOCD finding was positive, simply applies the recommended quantum (percentage) of impairment to the calculation of the claimant's permanent disability pension and notifies the claimant of the award. The award may be retroactive if, in the opinion of the ACOCD, the claimant showed signs of having had an asbestotic condition for some time. Of course, in such a case, the claimant's quantum of impairment would also be likely to have been set by the ACOCD at a higher than minimum level.[48]

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[42] RCA Transcript, Evidence of Dr. Jerome J. Vingilis, 8 June 1982, Volume no. 39, p. 113.

[43] RCA Transcript, Evidence of Dr. Cameron C. Gray, 16 July 1982, Volume no. 51, pp. 38–39.

[44] In testimony, the Board's Chest Disease Consultant cited a quantum of 35% as a possibility which, however, he could not recall explicitly. See RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, p. 53. Professor Barth's study explicitly recorded one claim in which the quantum was 75%. See Barth, *Workers' Compensation and Asbestos in Ontario,* p. 3.10.

[45] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, p. 51.

[46] RCA Exhibit II-54(b), in RCA Transcript, Evidence of Dr. Jerome J. Vingilis, 8 June 1982, Volume no. 39, p. 51: Assorted Forms.

[47] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, pp. 130–131. With respect to the reasons given for ACOCD findings, Dr. Jerome J. Vingilis, a member of the ACOCD, affirmed in testimony that the following would be a "typical" example of a negative ACOCD report:
"No radiographic progression seen.
Pulmonary function tests are within normal limits.
They have not deteriorated since the last examination.
Signs of asbestos dust inhalation, but no significant asbestosis.
Suspect angina. Re-examine in two years.
We recommend that the claim not be allowed." RCA Transcript, Evidence of Dr. Jerome J. Vingilis, 8 June 1982, Volume no. 39, p. 114.

[48] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, pp. 147–149.

Professor Barth's data indicated that 282 of the 391 asbestosis claims initiated from 1970 through 1980 reached the ACOCD. Of these 282 claims, 200 were affirmed by the ACOCD, and the remaining 82 were the subject of negative findings.[49] A claim that receives a negative finding, like all Board claims which are subject to denial, must be forwarded by the Claims Adjudication Branch to the Claims Review Branch before the claimant is notified. In the recollection of the Board's Chest Disease Consultant, the Claims Review Branch has never questioned a negative finding by the ACOCD.[50] As for the claims concerning which the ACOCD has determined a quantum of impairment, the percentage does not come before the Claims Review Branch because the claim has been deemed successful and hence has been awarded by the Claims Adjudication Branch.

We have been unable to procure any statistical evidence concerning the number of asbestosis claims that have been appealed because the claimant was either denied or dissatisfied with the quantum of impairment he received. However, precisely because asbestosis is an irreversible and normally progressive disease, the claimant who has received a partial disability pension will be reviewed regularly by the ACOCD; so, for that matter, may a claimant who was denied because he was initially rated by the ACOCD at zero impairment. We note that to facilitate re-examination, the physician members of the ACOCD regularly travel to geographic locations outside Toronto.[51] On the basis of data covering the years 1970 through 1980, Professor Barth found that 57 claimants, all of whose claims were initiated between 1970 and 1976, were examined by the ACOCD on five or more occasions.[52] Again, according to Professor Barth, recipients of partial disability pensions most commonly received one change in their impairment rating over a seven- or eight-year period.[53] Finally, Professor Barth estimated that about 30% of the individuals initially rated by the ACOCD at zero impairment ultimately received compensation.[54]

This last point adds significance to the initial gatekeeper role of the Board physician vis à vis the ACOCD. We take it that a claim not seen by the ACOCD cannot be scheduled for re-examination by the ACOCD as a matter of course. In a very real sense, an asbestosis claim seen once by the ACOCD is never considered closed so long as the claimant lives.

The role of the Board physician as gatekeeper to the ACOCD lies merely at the surface of some deep concerns we have developed on the subject of asbestosis claims. We find an absence of any coherent Board policy to guide the adjudication of such claims. This is as true of the manner in which the ACOCD is left to determine the matter of impairment as it is of the procedures through which asbestosis claims are handled. Let us first address the matter of impairment.

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[49] Barth, *Workers' Compensation and Asbestos in Ontario*, p. 6.27.

[50] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, p. 56.

[51] Ibid., p. 44.

[52] Barth, *Workers' Compensation and Asbestos in Ontario*, pp. 6.13, 6.15.

[53] Ibid., pp. 6.16, 6.21.

[54] Ibid., p. 6.10.

B.3 Determining the Quantum of Impairment

No written guideline or policy exists to guide or structure the deliberations of the ACOCD. In the words of the Board's Chest Disease Consultant: "We have been sending files to the Committee for fifty-five years, and when we send a claim to the Committee for asbestos [sic], they know what we want. They know that we want a diagnosis, and we want an estimate of the impairment or the disability that arises from that."[55]

The absence of guidance is carried to the point where the Corporate Board's benefit of doubt policy, which as we have seen is otherwise applicable to asbestos-related disease claims, does not extend to the ACOCD. The Board's Vice-Chairman of Administration expressed surprise that this should be so,[56] but it was confirmed as a fact by Dr. Gray, consultant to the ACOCD,[57] and by the Executive Director of the Board's Medical Services Division, Dr. William J. McCracken.[58] The latter went on to explain in his testimony before us that this fact is not accidental. In his opinion, ". . . benefit of doubt should not be used in the medical evaluation of the problem," but instead is a tool pertinent only to the administrative or legal aspects of the claims process.[59]

If there is a rationale for this state of affairs, it can be found in the ACOCD's practice of re-examining claimants at more or less frequent intervals. As Dr. Gray stated in testimony:

> I have a recollection of the odd [case] where there was some real doubt, and it was suggested that the man be seen at an earlier stage than he would have been otherwise. Instead of having a two-year appointment, he was given a one-year appointment or he was given a six-month appointment, because there might have been some doubt about change and therefore it

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[55] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, pp. 34–35.

[56] RCA Transcript, Evidence of Mr. Alan G. MacDonald, 11 August 1982, Volume no. 55, p. 77.

[57] RCA Transcript, Evidence of Dr. Cameron C. Gray, 16 July 1982, Volume no. 51, p. 70.

[58] RCA Transcript, Evidence of Dr. William J. McCracken, 20 July 1982, Volume no. 53, p. 69.

[59] Ibid., pp. 99–100.

would be wise to look at him again, and maybe a little earlier than usual.[60]

This rationale, whatever its merit, does not alter the fact that the Board, either at the Corporate level or through its staff physicians, has never chosen to guide or structure the discretion of the ACOCD. This Committee, to be sure, is composed of expert and knowledgeable physicians. A number of factors therefore come into play as *internal* checks on its discretion. There is the check provided by professional expertise and long experience: members have often been selected from the Chest Surveillance Programme because of the familiarity they have already gained with assessing early signs of chest disease. There is the check provided by group deliberation in the presence of the consultant specialists who sit on the Committee. Then there is the medical literature with which the members of the Committee are thoroughly familiar.

A member of the ACOCD, Dr. Jerome J. Vingilis, shared with us during testimony an example of the professional literature to which the ACOCD adheres closely.[61] This is the textbook entitled *Guides to the Evaluation of Permanent Impairment* prepared by the Committee on Rating of Mental and Physical Impairment of the American Medical Association (AMA). We reproduce from this book in Table 13.1 a succinct summary of classes for respiratory impairment. Under each progressively more severe class of impairment, the table describes the corresponding roentgenogram (x-ray) appearance, degree of dyspnea (shortness of breath), tests of ventilatory function, and arterial oxygen saturation (oxygen uptake).

Material submitted to us by the Board subsequent to our hearings has yielded another illustration of the ACOCD approach to the measurement of impairment.[62] We reproduce in Table 13.2 a worksheet prepared by Dr. Jaan O. Roos of the ACOCD. Up the left-hand vertical scale of the worksheet is a rating from 0 to 4 of classes of progressive impairment. The horizontal scale presents four different evaluation divisions — Physiologic, Anatomic, Clinical, and Exposure — yielding the acronym PACE. In his written commentary that accompanied the PACE diagram,[63] Dr. Roos

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[60] RCA Transcript, Evidence of Dr. Cameron C. Gray, 16 July 1982, Volume no. 51, p. 71.

[61] RCA Exhibit II-54(a), in RCA Transcript, Evidence of Dr. Jerome J. Vingilis, 8 June 1982, Volume no. 39, p. 51: American Medical Association, Committee on Rating of Mental and Physical Impairment, *Guides to the Evaluation of Permanent Impairment* (Chicago: AMA, 1971), excerpts.

[62] Letter and attachments from Dr. Charles Stewart, Chest Disease Consultant, Medical Branch, Medical Services Division, Workmen's Compensation Board to the Royal Commission on Asbestos, 2 November 1982.

[63] Dr. Jaan O. Roos, Member of the ACOCD, "Advisory Committee on Occupational Lung Disease," undated, 11 pages, attached to letter from Dr. Charles Stewart to the Royal Commission on Asbestos, 2 November 1982.

Table 13.1

Classes of Respiratory Impairment

	Class 1 0% Impairment	Class 2 10%-20% Impairment	Class 3 25%-35% Impairment	Class 4 50%-70% Impairment
Roentgenogram appearance	Usually normal but there may be evidence of healed or inactive chest disease including, for example, minimal nodular silicosis or pleural scars.	May be normal or abnormal.	May be normal but usually is not.	Usually is abnormal.
Dyspnea	When it occurs, is consistent with the circumstances of activity.	Does not occur at rest and seldom occurs during the performance of the usual activities of daily living. The patient can keep pace with persons of same age and body build on the level without breathlessness, but not on hills or stairs.	Does not occur at rest but does occur during the usual activities of daily living. However, the patient can walk a mile at his own pace without dyspnea although he cannot keep pace on the level with others of the same age and body build.	Occurs during such activities as climbing one flight of stairs or walking 100 yards on the level, on less exertion, or even at rest.
Tests of ventilatory function (At least two should be performed.)				
$FEV_{1.0}$ FVC MVV	Not less than 85% of predicted.	70%-85% of predicted.	55%-70% of predicted.	Less than 55% of predicted.
Arterial oxygen saturation (when performed)	Not applicable.	Not applicable.	Usually 88% or greater at rest and after exercise.	Usually less than 88% at rest and after exercise.

SOURCE: American Medical Association, Committee on Rating of Mental and Physical Impairment, *Guides to the Evaluation of Permanent Impairment* (Chicago: AMA, 1971), Table 8, p. 75.

noted that the normal clinical derivation of the data moves from right to left; that is, from Exposure (occupational, smoking, and activity) to Clinical (patient history and physical examination) to Anatomic (x-ray and biopsy) to Physiologic (pulmonary findings obtained at rest and derived from exercise studies). He observed that somewhat more weight is assigned by the Committee to Anatomic and Physiologic evidence because these categories are more specific and subject to objective measurement. Dr. Roos supplied an illustrative summary of the findings that generally correspond to the classes of progressive impairment of from 0 to 4 for each of the categories of clinical evidence. This is reproduced as Table 13.3. With respect to what the numerical classes of impairment are intended to convey, we note the recurring presence under the Physiologic and Anatomic columns of the words "slight," "moderate," "severe," and "extremely severe" or "extreme." Returning to Table 13.2, we note as well that the right-hand vertical scale of the PACE worksheet assigns two or more 10-point ranges of percentage disability to each numerical class of impairment.

Dr. Roos emphasized that the PACE diagram is ". . . not used at all in routine practice or evaluations," nor is it ". . . intended . . . in a mechanically arithmetic sense with the summation of points to derive a specific disability rating."[64] Instead, it is meant to ". . . serve as a teaching aid and an illustration of the type of thought which is used in disability assessment by this Committee."[65] We accept Dr. Roos' valuable illustration in the spirit in which it is intended and indeed as enlighteningly indicative of the enormous task which is assigned to the collective judgement of the ACOCD without the benefit of Board guidance.

To appreciate the full magnitude of the task performed by the ACOCD, one can begin with the uncertainty that shrouds clinical evidence. Dyspnea (shortness of breath) is, like pain, heavily influenced by the subjective feelings of the patient. The extent to which the reading of x-rays is associated with large variations of interpretation among different readers, and even on the part of the same reader, is uncontested.[66] In addition, developments that are producing new imaging techniques offer a potent reminder that the limits of x-ray diagnosis are technologically determined and may be particularly insensitive to determining the early fibrotic changes due to asbestosis.[67] As for pulmonary function tests, Dr. Annalee Yassi, in

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[64] Ibid., p. 7.

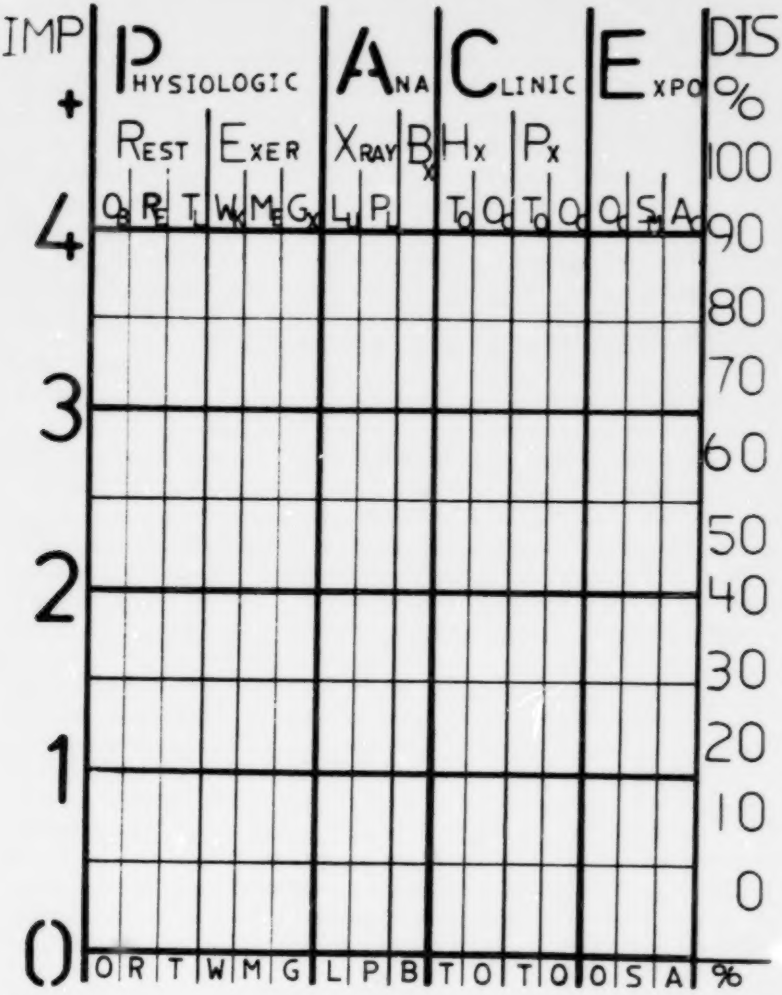
[65] Ibid., p. 8.

[66] See, for example, R.B. Reger and W.K.C. Morgan, "On the Factors Influencing Consistency in the Radiological Diagnosis of Pneumoconiosis," *American Review of Respiratory Disease* 102 (1970): 905–915; and J. Yerushalmy, "The Statistical Assessment of the Variability in Observer Perception on Roentgenographic Pulmonary Shadows," *Radiologic Clinics of North America* 7:3 (December 1969): 381–392.

[67] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, pp. 19–21; and RCA Transcript, Evidence of Dr. William J. McCracken, 20 July 1982, Volume no. 53, pp. 71–73.

Table 13.2

Worksheet Illustration of ACOCD Approach to Impairment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EXPO
DIS
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OB RE TL WK ME GX LU PL TO OC TO OC OC SM AC
O R T W M G L P B T O T O O S A %

Name .... **X-** .... **Date** .... **Place** ....

Table 13.2 (continued)

Worksheet Illustration of ACOCD Approach to Impairment

Notes:

IMP = Impairment.
DIS = Disability rating.
EXER = Exercise.
Bx = Biopsy (lung or pleura).
Hx = History.
Px = Physical examination.
ANA = Anatomic.
CLINIC = Clinical.
EXPO = Exposure to hazard.
OB = Obstructive ventilatory defect.
RE = Restrictive (non-obstructive) ventilatory defect.
TL = Transfer Factor for CO (Diffusing Capacity).
WK = Work output.
ME = Lung mechanics limitation to exercise performance.
GX = Gas exchange limitation to exercise performance.
LU = Lung.
PL = Pleura.
TO = Total rating of abnormality (by history or physical examination).
OC = 1. Occupational component of above.
2. Occupational respiratory hazard.
SM = Smoking history.
AC = Current general physical activity level.

4+ = The most severe possible hazard, abnormality, or finding.
3+ = Severe hazard, abnormality, or finding.
2+ = Moderate hazard, abnormality, or finding.
1+ = Slight hazard, abnormality, or finding.
0+ = No significant hazard, abnormality, or finding.

SOURCE: Dr. Jaan O. Roos, Member of the ACOCD, attached to letter from Dr. Charles Stewart to the Royal Commission on Asbestos, 2 November 1982.

Table 13.3

Illustrative ACOCD Findings by Class of Impairment and Category of Evidence

Class of Impairment	Physiologic	Anatomic	Clinical	Exposure
4	(max.) Extremely severe occupationally relevant impairment	(max.) Category 3/4 pqrstu & or grade 3 pqrstu + C Ontario 6	(max.) Maximal symptoms at rest relevant to the occupational exposure	(max.) Very severe exposure in a well-known [hazard] for adequate duration
3	Severe impairment relevant to occupational exposure	Cat. 3 (ILO 80) *Extreme* fibrothorax cat C Ontario 6	Relevant symptoms at rest or minimal exertion	Adequate duration of exposure in a well-known hazard which has constantly produced pneumoconiosis
2	Moderate impairment relevant to occupational exposure	Cat. 2 (ILO 80) Cat A or B severe fibrothorax Ontario 5-6	Relevant symptoms on slight exertion	Adequate exposure duration in a known but uncommon hazard for fibrogenesis
1	Slight but relevant impairment for occupational exposure	Cat. 1 (ILO 80) Moderate pleural thickening &/or pleural calcification Ontario 4	Relevant symptoms on moderate exertion	Adequate exposure duration in a probable but uncommon hazard situation
1/2	Borderline but relevant impairment	Cat. 1 (ILO 80) slight but definite pleural thickening Ontario 4	Relevant or reasonable symptoms on marked exertion only	Some exposure but doubtful duration of source

SOURCE: Dr. Jaan O. Roos, Member of the ACOCD, attached to letter from Dr. Charles Stewart to the Royal Commission on Asbestos, 2 November 1982.

her study for the Weiler inquiry, noted such potentially distorting effects as variations in effort exerted by different patients and differing standards of what constitutes "normal" function or for that matter "mild," "moderate," or "severe" impairment or disability.[68] The ACOCD, amidst all these sources of uncertainty, does more than distinguish among claimants in accordance with categories of impairment like "mild" or "moderate"; it further distinguishes within categories of impairment by recommending different percentages of disability.

The very terms "impairment" and "disability" in turn complicate the task of the ACOCD. Thus far in this chapter we have used these terms interchangeably, in part for the sake of simplicity, in part because the terms exhibit different degrees of overlap both in the statutory language of the *Workers' Compensation Act* and in medical practice.

Statutory eligibility for a permanent disability pension is prescribed by section 43(1) of the Act in the following words: "Where permanent disability results from the injury, the impairment of earning capacity of the employee shall be estimated from the nature and degree of the injury. . . ."[69] The effect of this language, as we understand it, is to define by statute two words, "disability" and "impairment," each of which is made a matter of medical determination, in terms of the other. Clearly, whether or not "permanent disability results from the injury" calls for medical determination. Just as clearly, the words "the impairment of earning capacity of the employee" are drained of any socio-economic connotations by the stipulation that this impairment "shall be estimated from the nature and degree of the injury." This is again a matter for medical determination. From this we conclude that the requirements of section 43(1) make disability and impairment synonymous and matters for medical determination.

In performing its task, which is ultimately governed by the language of section 43(1), the ACOCD must live with the fact that the medical literature offers quite different versions of the words "impairment" and "disability." According to the AMA *Guides to the Evaluation of Permanent Impairment,* to which we have already had occasion to refer, permanent impairment is ". . . a purely medical condition." It is ". . . any anatomic or functional abnormality or loss after maximal medical rehabilitation has been achieved. . . ."[70] For its part, permanent disability ". . . is not a purely medical condition. A patient is permanently 'disabled' or 'under a permanent disability' when his actual or presumed ability to engage in gainful activity is reduced or absent because of 'impairment' which, in turn, may or may not be combined with other factors."[71]

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[68] Yassi, "Occupational Disease and Workers' Compensation in Ontario: A Report for the Weiler Inquiry," chap. 3, sec. 4.3.

[69] R.S.O. 1980, c. 539, s. 43(1).

How has the ACOCD resolved the difference between the statute and the medical literature? With one notable exception, our answer to this question is that the ACOCD has opted for the statutory approach that makes impairment and disability synonymous. In making this finding, we pronounce ourselves in basic agreement with the observations of the Board's Chest Disease Consultant, Dr. Charles Stewart,[72] and cite the following evidence.

First, there is the matter of cigarette smoking in relation to asbestosis. As we know from our review of the health literature in Chapter 5, cigarette smoking cannot initiate the fibrotic process that leads to asbestosis. On the other hand, however, the deviation from normal of the lung functions of a victim of asbestosis is likely greater if the victim is a smoker. This increases the difficulty of assessing impairment caused by asbestosis. The ACOCD resolves this difficulty by ignoring smoking habits when it recommends quantum of impairment. If the claimant, in the words of Dr. Vingilis, ". . . has simple, chronic, obstructive lung disease, cigarette-related, we do not discriminate. . . ."[73]

Second, it is acknowledged that lung function tests, particularly exercise tests, measure work-related disability as well as impairment. Thus, a particular lung function measurement may indicate that an individual could do average factory work but not heavy construction work. Even though the ACOCD may know that a given individual is not disabled in relation to the work associated with his current employment, it will equate that individual's impairment with disability.[74]

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[70] American Medical Association, Committee on Rating of Mental and Physical Impairment, *Guides to the Evaluation of Permanent Impairment*, p. iii. We note a comparable but lung-specific definition of impairment by the Canadian Thoracic Society and the Health Standards Division of the Department of National Health and Welfare: "A reduction of lung function as compared with predicted so-called 'normal values.' " Health and Welfare Canada, *Task Force on Occupational Respiratory Disease (Pneumoconiosis)* (Ottawa: Minister of National Health and Welfare, February 1979), p. 7. Dr. Cameron C. Gray of the ACOCD served as a member of this Task Force.

[71] American Medical Association, Committee on Rating of Mental and Physical Impairment, *Guides to the Evaluation of Permanent Impairment*, p. iii. The definition of disability by the Canadian Task Force is: "The inability of an individual to perform his/her usual pattern of activities at work, at home, and in his/her leisure time." Health and Welfare Canada, *Task Force on Occupational Respiratory Disease (Pneumoconiosis)*, p. 7.

[72] Letter from Dr. Charles Stewart to the Royal Commission on Asbestos, 2 November 1982.

[73] RCA Transcript, Evidence of Dr. Jerome J. Vingilis, 8 June 1982, Volume no. 39, p. 54.

There is, however, a noteworthy exception to our finding that the ACOCD equates impairment and disability: it arises in cases of severe to extremely severe asbestosis. In its advanced stages, asbestosis progresses from a state where it precludes stair-climbing to one where it confines an individual to his home and then makes him virtually bedridden. At these stages, the individual is, to all intent, totally disabled from work and from every semblance of the activities of day-to-day life. But is that individual's impairment as high as his disability? There will be situations where the ACOCD cannot tell on the basis of its examinations because the individual, in the words of Dr. Gray, is ". . . too ill, too sick, too disabled to come."[75] Such an individual may be awarded temporary disability benefits by the Board because of his bedridden condition, but his permanent disability status, for lack of opportunity to ascertain whether his impairment was occasioned by asbestosis, is left in limbo. After the death of that individual, it will become the task of the Board, with the advice of the ACOCD, to determine retrospectively, perhaps with the aid of pathological evidence from an autopsy, whether the individual was 100% impaired. There is thus an incongruence between disability and impairment in the more advanced stages of asbestosis. We propose to address this state of affairs presently, but by redefining impairment rather than insisting that it be aligned with disability.

Before doing so, however, let us summarize what has been said so far by saying that with respect to determining the quantum of impairment, the ACOCD has: (i) shouldered the burden of an enormous task fraught with medical uncertainty; (ii) translated categories of impairment such as mild, moderate, severe, and extreme into more refined percentage categories of impairment/disability; (iii) basically viewed impairment and disability, in line with the Act, as synonymous; and (iv) accomplished all of this without guidance from the Board.

The first strong conviction we now express is that the absence of Board guidance in the matter of compensation for asbestosis must come to an end. The same considerations that motivate our call for clear and publicly known eligibility rules in Chapter 12 are applicable to the specific task of determining the quantum of impairment caused by asbestosis. Both for the sake of certainty and simplicity in claims adjudication, and to serve horizontal equity by promoting the similar treatment of similarly impaired individuals, classes of impairment should be explicit as a matter of Board policy.

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[74] Ibid., p. 49.

[75] RCA Transcript, Evidence of Dr. Cameron C. Gray, 16 July 1982, Volume no. 51, p. 49.

As for what constitutes impairment, we believe that the definition should be grounded in medical considerations for both conceptual and pragmatic reasons. Conceptually, defining impairment as a medical condition will retain consistency with the medical literature. Pragmatically, defining impairment as a medical condition will be consistent either with a statutory approach which, like the present one, confounds impairment with disability or with a statutory approach that would seek to distinguish impairment from socio-economic disability.

We begin with the definition of impairment, cited above, from the AMA *Guides*, as "any anatomic or functional abnormality or loss after maximal medical rehabilitation has been achieved." Because asbestosis, sadly, is irreversible, the person in whom it is diagnosed is irremediably beyond "maximal medical rehabilitation." The part of the AMA definition which remains operationally applicable to asbestosis is therefore "any anatomic or functional abnormality or loss." Impairment is therefore narrowly defined in *physical* terms.

But medicine embraces psychological as well as physical impairment. The individual who is told that he has asbestosis learns that he suffers from an irreversible and normally progressive disease. We are not aware of medical studies that deal specifically with psychological impairment that might arise from asbestosis. There are studies, however, which document that adverse psychological effects are associated with medical conditions which have not even produced clinical symptoms, let alone permitted the diagnosis of irreversible disease. Thus, one study has found that individuals who are mislabelled as having high blood pressure show a significant decrease in their perception of well-being.[76] In another study, this one dealing with Canadian steelworkers, it was found that when previously unaware workers were told they had high blood pressure there was a rise in absenteeism in comparison to workers who were unaware of their blood pressure. The absenteeism rate has persisted over a number of years and the most recent analysis indicates that these workers are earning about $1,000 less per person per year than their matched cohorts.[77] Since there is an effective treatment for high blood pressure and this condition is not associated with a significant shortening of life expectancy if properly treated, the degree of attitudinal change in these otherwise healthy workers is of some significance. More serious life events can reasonably be expected to coincide with more pronounced degrees of attitudinal change. There are studies documenting depression and suicide among victims of pulmonary and cardiorespiratory diseases.[78] In fact, and specifically in the realm of asbestos, one study of mortality patterns among factory workers who exhibited significant excess deaths from respiratory disease found 30 suicides where only 17 might have been expected, a statistically significant increase at the 1% level in the incidence of this tragic outcome of depression.[79]

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[76] R. Brian Haynes et al., "Improvement of Medication Compliance in Uncontrolled Hypertension," *The Lancet* (12 June 1976): 1265–1268.

[77] David L. Sackett et al., "Randomised Clinical Trial of Strategies for Improving Medication Compliance in Primary Hypertension," *The Lancet* (31 May 1975): 1205–1207.

At the level of recognized medical textbooks, the adverse psychological impact of illness, injury, and loss is fully acknowledged. Dr. Mardi J. Horowitz of the Department of Psychiatry at the University of California at San Francisco has written as follows:

> Considering the general disparities of psychological research, a rather remarkable concordance is found in the clinical, field, and experimental studies of response to the stress of serious life events or to vicarious simulations of such events. The frequency of two broadly defined states increases after such occurrences; one is characterized by an unusual level of intrusive and emotional memories or fantasies, the other by qualities of emotional numbing and ideational avoidance or denial.[80]

Dr. Horowitz went on to say:

> Insofar as their severity is consonant with the meaning of these events to the person, such states of denial or intrusion are normal. Pathological states are those that are unusually intense and, at the same time, are maladaptive to the person.[81]

We have learned that the Workers' Compensation Board is neither unaware of, nor insensitive to, the adverse psychological consequences that can afflict claimants *in the realm of accidents.* On February 9, 1982, the Corporate Board approved a guideline entitled "The Adjudication of Claims for Psychotraumatic Disability," whose full text is reproduced in the Appendix to this chapter. This document opens with the words: "An employee is entitled to benefits when personal injury is sustained through an accident which arises out of and occurs in the course of employment. 'Injury' includes both physical and emotional disability." It goes on to say that, "Psychotraumatic disability is considered to be a temporary condition." Subsequently, the guideline stipulates that one of the criteria for entitlement is fulfilled when "[t]he psychotraumatic disability is shown to be related to extended disablement and to non-medical, socio-economic factors, the majority of which can be directly and clearly related to the compensable injury."

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[78] Kent Smith, "Psychological Implications of Pulmonary Disease," *Clinical Notes on Respiratory Disease* 16 (1977): 3–11; I. Sakinofsky, "Depression and Suicide in the Disabled," in *Behavioural Problems in the Disabled,* ed. D.S. Bishop (Baltimore, Maryland: Williams and Wilkins, 1980), chap. 2, pp. 16–51; and N.L. Farberow et al., "Suicide Among Patients with Cardiorespiratory Illness," *Journal of the American Medical Association* 195:6 (7 February 1966): 128–134.

[79] Cynthia Robinson, Richard A. Lemen, and Joseph K. Wagoner, "Mortality Patterns, 1940–1975 Among Workers Employed in an Asbestos Textile, Friction and Packing Products Manufacturing Facility," in *Dusts and Disease,* eds. Richard A. Lemen and John M. Dement (Park Forest South, Illinois: Pathotox Publishers Inc., 1979), p. 134.

[80] Mardi J. Horowitz, "Psychological Processes Induced by Illness, Injury and Loss," in *Handbook of Clinical Health Psychology,* eds. T. Millon, C. Green, and R. Meajher (New York: Plenum Press, 1982), p. 53.

[81] Ibid., p. 54.

What is clear from this is that the Board: (i) has declared a policy of compensating for adverse psychological conditions that arise from work-related accidents; (ii) views the compensation as involving temporary benefits; and (iii) associates the adverse psychological conditions with socio-economic disability rather than medical impairment. With respect to the last point, the Board appears to us to be consistent with its statute because the Act confounds impairment with disability only with respect to permanent disability, not with respect to temporary disability. The Executive Director of the Board's Medical Services Division confirmed, at a meeting with Commissioner Mustard and our Legal Counsel,[82] that Board practice indeed coincides with the above characterization. He could not think of an asbestosis case where an allowance for psychological impairment had been made; neither could the Board's Chest Disease Consultant in his testimony before us.[83]

If the matter of impairment arising from asbestosis has remained grounded solely in physical considerations, this is occasioned only in part by the fact that the Board has not addressed the psychological impact of disease. The AMA *Guides*, to which the ACOCD adheres, offers only a physical definition of impairment. That this should be so is broadly consistent with the widely accepted professional notion that medicine, with greater or lesser allowance for uncertainty, can measure physical impairment more readily than it can measure psychological impairment. This does not deny the medical content of psychological impairment; even less does it deny the reality of the psychological impact of a diagnosis of asbestosis on a claimant. It is instead an assertion of what medicine confidently considers more readily measurable.

In our judgement, what is measurable as medical impairment and what is compensable as medical impairment need not and should not be synonymous. What is medically compensable should embrace matters which: (i) can be grounded in medical fact; and (ii) can be related to medical impairment in the realm of adjudication.

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[82] RCA Meeting with Dr. J. Fraser Mustard, Mr. John I. Laskin, and Dr. William J. McCracken, 1 December 1982.

[83] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, pp. 134–135.

We believe that we can reasonably infer that a phenomenon is a medical fact if, whether or not it is quantifiable, it is recognized as such in the professional literature. The authorities we have cited satisfy us that as a matter of general medical fact, psychological impairment can be expected to result from learning that one suffers from an irreversible, normally progressive disease. We can then find stronger and more specific reason for inferring that psychological impairment arises as a matter of medical fact if, in the circumstances just outlined, experienced practitioners of clinical medicine have recognized its reality in the patients they treat. On this score, we cite the following evidence from our transcript of sworn testimony, wherein Mr. Nick McCombie, representing the Injured Workers' Consultants, was cross-examining Dr. Vingilis, member of the ACOCD:

> Q. . . . do you know if anyone has done any studies on the psychological impact of asbestos, insofar as a worker all of a sudden discovering that they do have an asbestos-related disease, and . . . the effect that that may or may not have on the individual?
>
> A. You notice this very much by examining those people. Yes.
>
> Q. You notice it, but do you know if there are any studies that are done by the Ministry or anyone else?
>
> A. I don't think there was psychological studies done, but that was a fact I felt very strongly about — many people been disturbed, and disturbed to depression and anxiety, and so on.[84]

We conclude that psychological impairment in asbestosis sufferers can be taken to be a matter of medical fact.

Can this medical fact of psychological impairment be related to medical impairment in the realm of adjudication? The authorities we have consulted involve cases of tort liability in which courts have made awards under the broad heading of non-pecuniary loss. Awards on grounds of non-pecuniary loss are the ones relevant to medical impairment because, as the term "non-pecuniary loss" suggests, such awards are not related to socio-economic disability. A review of a number of authorities dealing with personal injury claims makes it clear that the tort liability system recognizes and compensates for psychological impairment when it results from the defendant's wrongful act. It has been common for courts to compensate for severe and continuing pain even when clinical evidence discloses no organic cause for such pain.[85] Compensation has also been awarded for changes which an accident may have brought about in a victim. In making such an award that recognized the emotional impact on a victim of a serious scar on her leg, Chief Justice Gale of the Ontario Court of Appeal wrote:

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[84] RCA Transcript, Evidence of Dr. Jerome J. Vingilis, 8 June 1982, Volume no. 39, pp. 150–151.

> In our view, it is not necessary to experience a traumatic neurosis in order to obtain an award of damages for an emotional shock. . . . Nor is it absolutely necessary, although preferable of course, that psychiatric evidence be called.[86]

The courts are well aware that psychological states such as depression may not be objectively measurable. Indeed, courts have had to face the truly perplexing task of distinguishing the portion of depression that a plaintiff might have suffered from grief relating to the loss of a spouse in an accident, which at common law is not compensable, from the portion, which is compensable, of the plaintiff's depression occasioned by the accident-induced injuries which he himself suffered. In such a case, Mr. Justice Galligan of the Ontario High Court of Justice wrote:

> It is impossible to say, with any degree of precision whatsoever, what part the grief and sorrow play in the depression. I am satisfied that it is significant.
>
> The circumstances pose such difficulties in assessment that really, I think, have to go on the well-known principle that when one knows that damages have been sustained, the court must nevertheless assess them, even if doing so amounts to little more than making a guess.[87]

In the realm of court adjudication arising from tort liability cases, we have solid ground for finding that psychological impairment is viewed as a medical condition, and that difficulties of measurement, however intractable, are not a barrier in determining awards even if, to repeat Mr. Justice Galligan's phrase, "doing so amounts to little more than making a guess."

The same principle, in our view, should be applicable in the realm of workers' compensation. Let us always recall that the very rationale for such compensation rests on the fact that claimants have lost their common law rights to sue for damages. The Workers' Compensation Board, as we have noted, already provides, as a matter of policy, temporary benefits arising from psychotraumatic disability in accident victims. An interpretation of the British Columbia *Workmen's Compensation Act* provides an authority for finding that statutory words identical to those in the present Ontario Act permit the Board to compensate for psychological impairment in the medical sense of the word. Section 3(1) of the Ontario Act opens with the words:

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[85] *Canning v. McFarland and Gray*, [1954] O.W.N. 467; *Diederichs v. Metropolitan Stores Ltd.* (1956), 20 W.W.R. 246; *Krahn et al. v. Rawlings* (1977), 16 O.R. (2d) 166; *Hammer v. Mobile Oil of Canada Ltd. and Erick* (1979), 5 Sask. R. 296; *Russell v. Kostichuk* (1980), 15 C.C.L.T. 247.

[86] *Charters et al. v. Brunette et al.* (1973), 1 O.R. (2d) 131 at 132.

[87] *Montgomery v. Murphy et al.* (1982), 37 O.R. (2d) 631 at 639.

> Where in any employment, to which this Part applies, *personal injury* by accident arising out of and in the course of the employment is caused to an employee . . . . (Emphasis added.)[88]

Interpreting the words *personal injury* in a case in which a claimant with a permanent partial disability pension claimed that he was psychologically unfit for work, the British Columbia Compensation Commissioners wrote:

> Under the [B.C.] *Workmen's Compensation Act*, compensation is payable not for "injury" but for "personal injury." That is a technical term adopted by the legislature from the jargon of the common law courts. In those courts, that term includes psychological impairment as well as physical injury, and there is no ground for attaching a different meaning to the phrase in the present context.[89]

It is, therefore, apparent that the Ontario Board may indeed compensate for psychological impairment *qua* impairment under its current Act. Furthermore, we have reason for stating that, at least implicitly, the Board already recognizes such impairment in certain cases of permanent disability where, in line with the language of section 43(1), impairment and disability are synonymous. Thus, a claimant who is totally blinded by a workplace accident qualifies for a 100% permanent disability pension. Such a person retains his 100% disability/impairment award even though, with rehabilitation, the element of socio-economic disability has been reduced or perhaps even eliminated.

Over and above all the considerations we have dealt with thus far, we find a simple rationale grounded in common sense for recognizing the permanent psychological impairment of victims of irreversible and normally progressive disease as a matter of Board policy. In all but the most exceptional cases, the accident victim suffers his maximum loss at the time of the event. The worst that could happen has happened, and his condition is likely to be stabilized or even improved following rehabilitation. The victim of irreversible and normally progressive disease, for his part, must live with the notion that the worst has yet to happen: the likelihood of progressive physical impairment, of a shortened lifespan, and indeed of death from the disease or a related cause are his unsettling prospects. In our judgement these prospects, if anything, make the grounds for recognizing psychological impairment in victims of chronic, irreversible, lifespan-shortening disease more compelling than those that obtain in the realm of accidents. They also suggest that a policy of permanent compensation for such disease victims need not, of itself, dictate a change in the Board's psychotraumatic guideline that involves temporary compensation in individual cases that arise in the realm of accidents.

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[88] R.S.O. 1980, c. 539, s. 3(1).

[89] *Re the Determination of a Disability* (1973), 1 W.C.R. 19 at 24 (British Columbia Workmen's Compensation Commissioners).

We now address the question of how to compensate victims of asbestosis (and similar irreversible and normally progressive disease) as a practical matter. The answer, in our view, lies again in medical considerations. Psychological and physical impairment are both medical concepts; the difference between the two lies in the fact that physical impairment, within certain bounds dictated by uncertainty, is more readily quantitated than psychological impairment. It follows that, as a practical matter, a Board policy to compensate victims of asbestosis for their total impairment should be based upon a quantification of physical impairment, augmented by an automatic allowance for psychological impairment. Accordingly, such a policy should establish classes of physical impairment. The policy should then stipulate that the physicians of the Advisory Committee on Occupational Chest Diseases (ACOCD) have the task of recommending, on the basis of clinical examination, each claimant's class of physical impairment. The ACOCD should no longer be asked to determine a claimant's percentage of disability/impairment for compensation purposes within a particular class of impairment. Instead, corresponding percentages of total impairment, incorporating an allowance for psychological impairment, should be established by Board policy for each class of physical impairment. It would be left for the Claims Adjudication Branch to calculate each claimant's award in accordance with the Board-determined percentage of total impairment that corresponds to the class of physical impairment recommended by the ACOCD.

The Board's policy should recognize three classes of impairment with respect to the clinical recommendations asked of the ACOCD. On the basis of the material we have already reviewed, these should correspond to Classes 2, 3, and 4 of the AMA *Guides* regarding respiratory impairment. (See Table 13.1 above.) We note close similarities between these and the impairment Classes II, III, and IV depicted in the report of the Health and Welfare Canada Task Force on Occupational Respiratory Disease (Pneumoconiosis), which we reproduce in Table 13.4.

We take note of the terminology which the Health and Welfare Canada Task Force employed in describing impairment Classes II, III, and

Table 13.4

Classification of Impairment

	Class I	Class II		Class III		Class IV		Class V	
Dyspnea	(No impairment) The subject may or may not have dyspnea. If dyspnea is present it is for non-respiratory reasons.	(Mild Impairment) Dyspnea on fast walking on level ground or walking up a hill.		(Moderate Impairment) Dyspnea walking on level ground with person of the same age or walking one flight of stairs.		(Severe Impairment) Dyspnea after walking more than 100 meters at own pace on level ground.		(Very Severe Impairment) Dyspnea on talking or undressing or unable to leave his dwelling because of breathlessness.	
Pulmonary function tests		Obstructive	Restrictive	Obstructive	Restrictive	Obstructive	Restrictive	Obstructive	Restrictive
FVC	Normal*	N/A	More than 80% of Predicted Value	N/A	40%-80% of Predicted Value	N/A	Less than 40% of Predicted Value	Same as IV	Same as IV
FEV_1	Normal	More than 80% of Predicted Value	N/A	40%-80% of Predicted Value	N/A	Less than 40% of Predicted Value	N/A	Same as IV	Same as IV
DLCO	Normal	Normal or slightly abnormal	Less than 75% of Predicted Value	Usually more than 50% of Predicted Value	Usually more than 50% of Predicted Value	Reduced	Less than 40% of Predicted Value	Same as IV	Same as IV
Maximal VO_2	Normal more than 25ml/Kg/min**	Less than 25 ml/Kg/min**	Less than 25 ml/Kg/min	Less than 15 ml/Kg/min**	Less than 15 ml/Kg/min**	Less than 7 ml/Kg/min	Less than 7 ml/Kg/min	Less than 7 ml/Kg/min	Less than 7 ml/Kg/min
Arterial blood gases at rest	Normal	Normal	Normal	PO_2 more than 60 mmHg PCO_2 less than 45 mmHg	PO_2 more than 60 mmHg PCO_2 less than 45 mmHg	PO_2 more than 60 mmHg PCO_2 less than 45 mmHg	PO_2 more than 60 mmHg PCO_2 less than 45 mmHg	PO_2 less than 50 mmHg PCO_2 less than 45 mmHg	PO_2 less than 50 mmHg PCO_2 variable

Notes: *A normal value lies within 2 standard deviations of the mean. The mean value is standardized for age, sex, and height when applicable.
**If less, this limitation is secondary to non-pulmonary reasons.

SOURCE: Health and Welfare Canada, *Task Force on Occupational Respiratory Disease (Pneumoconiosis)* (Ottawa: Minister of National Health and Welfare, February 1979), p. 74.

IV and also of the descriptive terms used by Dr. Jaan O. Roos of the ACOCD in the written commentary that accompanied his PACE diagram. (See Table 13.2 above.) On the basis of these authorities, the three classes of physical impairment recognized by Board policy should be termed "Mild," "Moderate," and "Severe."

We explicitly reject the incorporation into Board policy of a class of "Extremely Severe" physical impairment which would correspond to Class V in the report of the Health and Welfare Canada Task Force. We have already noted that under prevailing ACOCD practice, asbestosis claimants in the most advanced stages of progression may be left to a *post mortem* determination of their physical impairment because they are too sick to be subjected to a clinical examination. We have further reason for rejecting the "Extremely Severe" class of physical impairment because, given the statutory ceiling of 100% for permanent disability, it would deny adequate recognition of psychological impairment. In this connection, it is necessary as well to recognize that individuals who have progressed to the "Severe" class of physical impairment can be said to bear an additional psychological burden because it is at this stage of progression that asbestosis is most likely to occasion death from related causes. Dr. Gray stated in testimony that usually ". . . the individual who dies of a right heart failure, the *cor pulmonale*, has significant and in fact severe impairment."[90] Dr. Gray also pointed out that the response of a person to treatment for bronchial pneumonia is likely to be ". . . less effective . . . because of the severe asbestosis. . . ."[91]

There remains the matter of setting, as a matter of policy, the percentage award for total medical impairment that should correspond to each class of physical impairment and that should incorporate an allowance for psychological impairment. Returning again to the classes taken from the AMA *Guides* and depicted in Table 13.1, we note that Class 2 is related to a range of 10 to 20% physical impairment; Class 3, to a range of 25 to 35%; and Class 4, to a range of 50 to 70%. With these ranges before us, and bearing in mind the wise observation of the courts that lack of objective measurement is no excuse for refraining from compensation based on judicious guesses, we determine that the following percentages, as a matter of Board policy, would fairly and equitably compensate asbestosis victims in each class of physical impairment for both physical and psychological impairment: Mild, 30%; Moderate, 60%; Severe, 100%.

In line with the above considerations, we recommend that:

13.1 The Workers' Compensation Board, by directive of the Corporate Board, should promulgate as a matter of Board policy a rule for

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[90] RCA Transcript, Evidence of Dr. Cameron C. Gray, 16 July 1982, Volume no. 51, p. 51.
[91] Ibid., p. 52.

determining the quantum of compensable physical and psychological impairment arising from asbestosis in claimants diagnosed by the Advisory Committee on Occupational Chest Diseases (ACOCD) as suffering from this disease. The rule should instruct the ACOCD to assign such claimants to one of three classes of physical impairment defined by competent medical authorities as corresponding to Mild, Moderate, and Severe respiratory impairment and direct that the percentages to be used by the Claims Adjudication Branch for calculating awards for physical and psychological impairment be:

(i) where the ACOCD finds Mild physical impairment, 30%;
(ii) where the ACOCD finds Moderate physical impairment, 60%; and
(iii) where the ACOCD finds Severe physical impairment, 100%.

We recognize that an asbestosis policy which incorporates an automatic allowance for psychological impairment has implications for other compensable diseases. We recognize as well that, unless these implications are confined within sensible boundaries, the matter of unravelling them could indefinitely postpone any recognition of psychological impairment in industrial disease victims. Accordingly, we offer two criteria to delineate the diseases whose compensation might include an automatic allowance for psychological impairment. First, such diseases should be workplace-specific, in the sense that their related industrial process is recognized as their necessary and sufficient cause. As a practical matter, the diseases that satisfy this criterion would be the diseases which, pursuant to our exposition in Chapter 12, qualify for Schedule 3 of the *Workers' Compensation Act*. Second, such diseases should involve, like asbestosis, chronic, irreversible disorders that shorten life expectancy. We recommend that:

13.2 In assessing the implications of Recommendation 13.1 for the compensation of other diseases, the Workers' Compensation Board should address those diseases whose associated industrial process is their necessary and sufficient cause and whose manifestation involves chronic, irreversible disorders that shorten the individual's life expectancy.

Under the current *Workers' Compensation Act*, the statutory provisions governing compensation for permanent disability make impairment and disability synonymous. We appreciate the fact that the *White Paper on the Workers' Compensation Act* envisages a markedly different approach to compensation. It proposes a dual compensation award system: one award to recognize impairment in its proper medical sense, the other to be paid in accordance with the victim's socio-economic disability. A dual award system has substantial implications for the cost and benefit levels of the entire scheme of workers' compensation in Ontario. Our deliberations have not embraced these implications because they are too vast, given our terms of reference and the nature of the submissions we have received, to be addressed by this Commission. The policy we have recommended for the compensation of asbestosis victims is confined to medical impairment; should a dual award system be instituted, this policy is applicable to the impairment award, not the socio-economic disability award.

We note, however, that this would not be possible under the language used in the legislative exposure draft that accompanies the *White Paper.* Section 1(1)(v) of this draft defines permanent impairment in conventional physical terms; indeed it repeats the words of the AMA *Guides* whereby permanent impairment is confined to mean "any anatomic or functional abnormality or loss after maximal medical rehabilitation has been achieved." We need not repeat the reasons for which we deem psychological impairment to be a valid component of permanent impairment in the medical sense of the term. It suffices to record our view that any future workers' compensation statute that seeks to define permanent impairment as a medical condition should do so in a manner that accommodates psychological impairment. Such a definition should be sensitive to the medical uncertainties that make it difficult to delineate the boundaries of psychological impairment and hence should be couched in language that gives the Board sufficient flexibility to set these boundaries as a matter of policy. To give a specific example, this would be permitted by a definition of permanent impairment that used words like the following: "any anatomic or functional abnormality or loss after maximal medical rehabilitation has been achieved and any psychological damage arising from such abnormality or loss that the Board may recognize as a matter of policy." We recommend that:

13.3 The statutory definition of impairment contained in the legislative exposure draft in the White Paper on the Workers' Compensation Act should be reviewed to ensure that the statutory language of any relevant amendment to the Workers' Compensation Act is couched in language that includes psychological impairment.

B.4 Restructuring the ACOCD

Now that we have addressed the substantive issues that are occasioned by the determination of quantum in asbestosis claims, what remain are matters of procedure. The first matter arises at the very threshold of the ACOCD: it is posed by the gatekeeper role of Board physicians, which permits them to recommend denial of asbestosis claims without referral to the ACOCD. Professor Barth expressed unease with this practice, and we share his concern. If we have reason to temper the degree of our own concern, it lies in the completely revised upper echelon of the appeals structure which we, in support of Professor Weiler, have recommended. Evidently, review by the new Appeals Tribunal, coupled with a Medical Review Panel, will give to the denied claimant the opportunity of an outside medical opinion as a check on the Board physician's finding that asbestosis cannot be diagnosed and that hence no referral is to be made to the ACOCD. However, we are mindful of the fact that this opportunity is extended to the claimant only at the final stage in the appeals process. Time has passed, and the extent to which a workers' compensation system should operate expeditiously revitalizes our concern.

The data from Professor Barth's study which we cited earlier in this chapter indicated that slightly more than one-quarter (109 of 391) of asbestosis claims were not referred to the ACOCD between 1970 and 1980. Let us observe that a claim not referred to the ACOCD is more than just denied and left to the appeals structure. Given the practice of the ACOCD, which we have noted, of rescheduling for early re-examination claimants it has seen but concerning whom it is in doubt, there is an advantage that is inherent to crossing the ACOCD threshold. Accordingly, we have considered recommending that the gatekeeper role of Board physicians be dispensed with altogether.

If we restrain ourselves from so recommending, it is from concern over the total workload of the ACOCD. The only figures we have on the Committee's workload have already been cited. They indicate some 400 to 450 chest disease (not just asbestosis) claims per year and may not take account of re-examinations. There is reason for being sensitive to the workload of the ACOCD to the extent that the timely disposition of the cases that *do* come to the Committee has its own relevance for the expeditious operation of the compensation system.

We choose to resolve the matter of the Board physician's role as gatekeeper to the ACOCD by proposing that discretion to deny access to the Committee be retained, but restricted to those claims which, in the stated opinion of a Board physician and subject to the usual check of the Claims Review Branch, are deemed frivolous. The term "frivolous" is well understood in law and indeed appears in the Ontario *Human Rights Code* as grounds for denying investigation of a complaint.[92] Claims not referred to the ACOCD on grounds of frivolity could still be appealed, and the Appeals Tribunal will doubtless have the competence to work out the basis of whether or not such grounds should be upheld in individual appeals. We therefore recommend that:

13.4 The Corporate Board should promulgate a rule applicable to the procedural adjudication of asbestosis claims which stipulates that a physician of the Medical Services Division is to refer such claims to the Advisory Committee on Occupational Chest Diseases unless, in the

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[92] *Human Rights Code, 1981*, S.O. 1981, c. 53, s. 33(1)(b).

> ***opinion of the physician subject to check by the Claims Review Branch, the claim is deemed frivolous.***

The second procedural matter we wish to address involves the *modus operandi* of the ACOCD itself. Our substantive recommendation on quantum of impairment leaves to the Committee the important tasks of diagnosing asbestosis and assigning claims to different classes of physical impairment. To be sure, the Committee's task will be simplified because only three classes of impairment are involved, and the Committee will be excused from its present duty of determining percentages of disability within classes of impairment. The Committee will therefore operate within bounds that are less vulnerable to medical uncertainty than is the case at present. But this is not to say that the Committee's role will in any way be trivialized. Cases at the borderline of the three impairment classes will remain problematic. More particularly, we have every expectation that the question of whether or not a claim falls into the Mild category of impairment will often continue to pose a major challenge to clinical judgement. We have already cited from Dr. Gray's testimony that disagreement within the ACOCD has been most likely to surface at low levels of impairment.

Several interconnected issues surround the *modus operandi* of the ACOCD. The first is its practice of leaving the clinical examination of a claimant to a single physician. The second is its practice of reaching consensus decisions with no record of dissenting votes. The third lies in the extent to which the Committee reviews its own work. In search of a standard of comparison, and following Professor Barth's lead, we have reviewed the arrangements through which the Commission de la santé et de la sécurité du travail du Québec diagnoses asbestosis and determines the quantum of impairment. There, as Professor Barth has pointed out, twelve physicians are allocated among four three-member panels.[93] Each panel is responsible for making the diagnosis and estimating the impairment of the claimants assigned to it. All three members of each panel are present for at least parts of each claimant's clinical examination. We find the enhanced degree of collective judgement made possible by this approach to clinical examinations attractive.

After we had completed our hearings, we asked the Quebec Commission whether it has procedures for recording dissents and for resolving these when they occur. We were informed by the Director of Medical Services of the Commission, Dr. Jean P. Lemieux, that precise procedures are in place.[94] The report on the medical evaluation of each claimant must be cosigned by all three members of the examining panel. Any dissenting opinion is recorded. Whenever a dissenting opinion occurs, the claimant's complete file is referred to a special committee composed of the chairmen of the four panels. The chairman of the panel from whom the dissenting opinion originated does not participate in the debate. The special committee's decision then becomes the decision of the primary level of adjudication and is subject to the normal channels of appeal.

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[93] Barth, *Workers' Compensation and Asbestos in Ontario,* p. 2.29.

[94] Letter from Dr. Jean R. Lemieux, Director, Medical Services, Commission de la santé et de la sécurité du travail du Québec to the Royal Commission on Asbestos, 25 October 1982.

Even allowing for the fact that the ACOCD schedules early re-examination of claimants concerning whom its opinion is divided, we find the Quebec procedures for recording and resolving dissents vastly superior to what prevails in Ontario. We note further that the Quebec procedure for resolving dissents provides a built-in review of the consistency of medical decisions.

On this last score, ACOCD re-examinations provide their own opportunity for the Committee to review its own work. We would not wish this practice to change; the re-examination of claimants is necessitated in any event by the fact that asbestosis is normally progressive. However, we observe that the Quebec *modus operandi* again beckons because, quite aside from the procedure for resolving dissents, all panels regularly meet to review each other's recent findings and decisions.[95]

Attracted as we are by the features of the Quebec system, we consider it important to address the issue of whether their transplantation to Ontario might generate excessive demands on the limited pool of relevant medical practitioners. Restructuring the ACOCD along the lines of the Quebec model would raise the Committee's membership from seven to twelve. Also to be borne in mind is the fact that an Ontario appeals system which incorporates Medical Review Panels will generate its own demands for physicians with similar expertise. The total numbers required, whether they amount to fifteen or twenty knowledgeable physicians, may appear small but even in a jurisdiction the size of Ontario, they may in fact be sizeable. Dr. Adam S. Little, in his review of Professor Barth's study, offered pointed reminders to this effect.[96] Diseases like asbestosis (and for that matter other diseases under the purview of the ACOCD), because they are industry-specific, afflict only a small proportion of the general population. It follows that such diseases will be seen by a correspondingly small proportion of practising physicians. In turn, a number of medical practitioners within this small proportion are likely to be company physicians whose positions as such may leave them perceived as unsuitable for compensation work.

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[95] Barth, *Workers' Compensation and Asbestos in Ontario*, p. 2.29.

[96] Reviews of studies prepared for the Royal Commission on Asbestos: Adam S. Little, "Critique re: *Workers' Compensation and Asbestos in Ontario*," Victoria, B.C., 1982, p. 5. (Mimeographed.)

That these are genuine limitations cannot, as a general proposition, be denied. They may indeed have been exacerbated by prevailing Ontario practices. The Workers' Compensation Board has relied heavily on the Chest Surveillance Programme as a source of ACOCD members in the past. This is in part because of the experience that Programme physicians have gained in reading x-rays. But Dr. Gray, in his testimony before us, affirmed that a physician need not have been involved in this Programme to gain the relevant experience.[97] In Dr. Gray's opinion, the pool of potential ACOCD members embraces:

> . . . a good many people who have expertise in lung disease and pulmonary disease, pulmonary function, pulmonary pathology, and I think with that group or that individual having an interest, and being put into such a thing as an advisory committee or some other group that is looking at occupational problems, he in time would develop an expertise.[98]

The Chest Surveillance Programme aside, Dr. Gray's opinion leads us to observe that the Board's accompanying practice of making ACOCD appointments tantamount to life terms has not abetted a dissemination of expertise among Ontario physicians. Moreover, this practice has yielded an ongoing situation where, as we estimate it, about half the current ACOCD membership hovers around retirement age. It follows that if Ontario has a shortage of physicians with experience in the realm of diagnosing industry-related pulmonary disease, that shortage is, at least in part, self-inflicted.

On the other hand, however, it is possible to approach the matter of Ontario's shortage, self-inflicted or otherwise, from a very different perspective. This is that the "shortage" rests upon an untenable notion of what constitutes the medical expertise necessary to diagnose pulmonary conditions that are industry-specific. At first blush, the notion that expertise should rest upon experience in diagnosing a quantity of these conditions appears reasonable. But it becomes increasingly untenable once it is placed in the context of the occupational health regime that now prevails in Ontario. Because they afflict a small subset of the general population, industry-specific diseases have always been exotic; the very thrust of the current Ontario occupational health regime, however, is to make these diseases extinct. For reasons given in Chapter 5, we have confidence that current levels of exposure will make asbestosis a disease of the past. It follows that over the next decade or two, the number of asbestosis cases will dwindle as inexorably as the number of workers who were exposed to the high dust levels of a decade or more ago. Physicians with any degree of quantitative experience in diagnosing asbestosis are therefore on their own inexorable path to extinction. This being so, it seems to us eminently reasonable to discount as increasingly untenable the notion that quantitative experience is a prerequisite for the expertise needed to diagnose the flow of compensation claims that remains. What is required instead is to seek out experienced physicians with broad diagnostic capabilities in the general run of pulmonary conditions and also physicians with perhaps less diagnostic experience but qualifications in occupational medicine. Such physicians should be sought out now, so that the specific experience possessed by the current senior members of the ACOCD can be imparted to them before it is dissipated by retirement. These current senior members, of course, are ideal candidates to chair the panels of a restructured ACOCD.

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[97] RCA Transcript, Evidence of Dr. Cameron C. Gray, 16 July 1982, Volume no. 51, p. 17.
[98] Ibid., p. 61.

Through what sources might their new colleagues be selected? The Quebec system, as Professor Barth pointed out, rests upon close liaison with the four medical schools of that province. In our view, the Board should actively cultivate Ontario's five faculties of medicine as a source of new members for a restructured ACOCD. Furthermore, practising respirologists with broad diagnostic experience have become expert in assessing pulmonary function regardless of underlying cause; they can be sought out by drawing on the co-operation of such professional bodies as the Canadian Thoracic Society and the Specialty Committee in Respiratory Medicine of the Royal College of Physicians and Surgeons of Canada.

In light of all of the above considerations, we recommend that:

13.5 The Corporate Board should increase the membership of the Advisory Committee on Occupational Chest Diseases (ACOCD) to not fewer than twelve physicians recommended by the Executive Director of the Board's Medical Services Division after consultation with appropriate university and professional bodies, and it should direct that the ACOCD be organized into not fewer than four subcommittees, each composed of three physicians including a subcommittee chairman designated by the Corporate Board.

We further recommend that:

13.6 The Corporate Board should issue the following procedural rules to conduct the operation of the Advisory Committee on Occupational Chest Diseases:

(i) Each subcommittee is to have the authority to diagnose claimants and assign claimants to classes of physical impairment.

(ii) The clinical examination of every claimant is to involve the full membership of the subcommittee to whom the claimant is assigned.

(iii) Every subcommittee report is to be signed by the full membership of the subcommittee, with any dissent recorded.

(iv) *In the event of subcommittee dissent, the relevant claim is to be reviewed by the chairmen of the three remaining subcommittees who shall then make the finding with respect to diagnosis and class of physical impairment.*

(v) *The entire membership of the Advisory Committee on Occupational Chest Diseases is to review the findings of its subcommittees on a periodic basis.*

We now address a third procedural matter: the degree of overlap which has existed between the Chest Surveillance Programme of the Ministry of Labour and the Advisory Committee on Occupational Chest Diseases. At one level, this overlap is to us only one symptom of the long-standing lack of Corporate Board guidance and indeed interest in the workings of the ACOCD. We feel bound to point out that in two separate submissions we received from the Board, the first its written submission,[99] the second its "procedural guidelines" supplied as an exhibit during the testimony of the Executive Director of its Claims Services Division,[100] the ACOCD has been referred to as the "Advisory Committee (A/C) of the Ministry of Labour."

More serious than such oversights is the annoyance that is aroused when claimants, particularly once they become privy to their files in the preparation of appeals, cannot distinguish what has come from the Chest Surveillance Programme and what is a finding of the ACOCD. Their confusion is compounded by the fact, mentioned earlier in this chapter, that the ACOCD writes its reports to the Board on Ministry of Labour stationery. The annoyance of confusion then becomes outright and justified indignation in those instances where a claimant finds he has been examined twice by the same physician but in different capacities unknown to the claimant: the first in the physician's capacity with the Chest Surveillance Programme, the second in the physician's different capacity as a member of the ACOCD.[101] For all this, the ACOCD has never been an organ of the Ministry of Labour and is well and truly a creature of the Workers' Compensation Board pursuant to section 71(3)(g) of the *Workers' Compensation Act*. The time to make this crystal clear is long overdue. For reasons that we will address momentarily, there is a single aspect of the overlap between the Ministry of Labour and the ACOCD that should continue: the facilities of the Medical Service Chest Clinic at 880 Bay Street in Toronto. Otherwise, we recommend that:

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[99] The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, p. 6.

[100] RCA Exhibit IV-1, The Workmen's Compensation Board, Ontario, Assorted Policy Statements and Forms.

[101] RCA Transcript, Submission by Mr. Ed Cauchi on behalf of the Asbestos Victims of Ontario, 28 January 1983, Volume no. 58, p. 116.

13.7 The Corporate Board should adopt a rule whereby physicians engaged in the work of the Occupational Health Branch of the Ministry of Labour are ineligible for membership on the Advisory Committee on Occupational Chest Diseases and should take measures to ensure that the secretariat of this Committee is supplied solely by the Workers' Compensation Board.

This much said, we believe that the Medical Service Chest Clinic facilities should continue to be shared by the Ministry of Labour and the ACOCD. Our reason is not historical but grounded in highly contemporary concerns. We recognize the value of the sequential x-rays produced by chest disease surveys to the diagnostic task of the ACOCD. We appreciate the fact that under the current designated substances regulations of the Ministry of Labour, new surveillance responsibilities fall on employers, but with the proviso that x-ray and pulmonary function facilities ". . . shall be engaged in a proficiency testing program with the [Ministry's] Occupational Health Branch."[102] In our view, it is of the utmost importance that the Government of Ontario facility with which employer facilities shall be engaged in proficiency testing should be the government facility devoted to purposes of both surveillance and compensation. It is of like importance that this government facility be periodically accredited by expert radiologists and respirologists with the same degree of thoroughness that pertains to hospital accreditations. We therefore recommend that:

13.8 The Occupational Health Branch of the Ministry of Labour and the Advisory Committee on Occupational Chest Diseases should continue to share the same x-ray and pulmonary function testing facilities, which should be accredited by an expert team of radiologists and respirologists at least every five years.

C. Compensating the Survivors of Deceased Asbestotics

C.1 The Current Setting

An asbestotic who is receiving a permanent disability pension from the Workers' Compensation Board is a person whose condition has been recognized by the Board as arising from the workplace and whose award is proportional to the degree of impairment which the Board has assessed as arising from asbestosis. The death of such an individual does not occasion a new claim for survivor benefits. Instead, the Board automatically considers whether such benefits are warranted.

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[102] Code for Medical Surveillance of Asbestos Exposed Workers, s. 4, in Regulation Respecting Asbestos, O. Reg. 570/82, made under the *Occupational Health and Safety Act*, R.S.O. 1980, c. 321.

This consideration begins at the moment that the Board, which has been mailing pension cheques to the individual concerned, becomes aware of the person's death, commonly by way of a telephone call.[103] Thereupon, the Claims Adjudication Branch takes the initial step of securing a copy of the death certificate to add to the already active claim file of the deceased. If the deceased was in receipt of a 100% permanent disability pension, survivor compensation is assured by section 43(7) of the *Workers' Compensation Act,* which mandates such compensation whatever the cause of death.[104] If, however, the deceased was in receipt of a *partial* disability pension, the Board is required to adjudicate whether or not survivor benefits are warranted. This requirement arises from the opening words of section 36(1) of the Act, which directs that survivor benefits flow, "Where death results from an injury." More specific still is the language of section 122(1), which stipulates that survivor benefits shall be allowed only if the victim's ". . . death is caused by an industrial disease and the disease is due to the nature of any employment in which he was engaged. . . ."

The deceased recipient of a partial disability pension for asbestosis was being compensated for a disease "due to the nature of [the] employment in which he was engaged." With respect to survivor benefits, the question for adjudication becomes: Was the death "caused" by this disease or by another disease which was also due to his employment? As we observe in Chapter 12, there is a noteworthy absence of any Board guideline or eligibility rule to structure the discretion of Board adjudicators with respect to the causality of death among asbestotics. To the extent that the death of such individuals arises from asbestos-related diseases other than asbestosis — namely, mesothelioma, lung, gastrointestinal, and laryngeal cancers — guidelines are available. But in the main, as we have come to understand it, Board procedure is to adjudicate the survivor claims of deceased asbestotics on a case-by-case basis.

Once the Claims Adjudication Branch has become aware of the partial disability pensioner's demise, it transfers the claim file to the Medical Services Division where, once again, the Board's Chest Disease Consultant, Dr. Stewart, or its Chest Disease Specialist, Dr. Dyer, takes charge. The file may contain, in addition to the death certificate, hospital records and the last attending physician's report.[105] Whatever the exact content of the file at the time it is received in the Medical Services Division, the Board physician will make what Professor Barth called "significant efforts" to obtain information.[106] This may include submissions from an attending specialist, the family physician, and — in instances where it has been performed — an autopsy report.

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[103] RCA Transcript, Evidence of Mr. John F. McDonald, 14 July 1982, Volume no. 49, pp. 141–142.

[104] R.S.O. 1980, c. 539, s. 43(7) states: "A dependant of an employee who was, at the time of his or her death, in receipt of an award for permanent disability which the Board has rated at 100 per cent or, but for his or her death, would have been in receipt of an award for permanent disability at the rate of 100 per cent is entitled to compensation as if the death of the employee had resulted from the compensable disability for which he or she received or would have received the permanent disability award."

[105] RCA Transcript, Evidence of Mr. John F. McDonald, 13 July 1982, Volume no. 48, p. 11.

In assessing the information at hand, the Board physician does not rely solely on his own expertise. Depending on the nature of the case, he will turn to any or all of three sources of specialized advice. These are Dr. Alexander C. Ritchie, the Board's consulting pathologist; Dr. Cameron C. Gray who, distinct from his membership on the ACOCD, is available as chest disease consultant to the Board; and the ACOCD itself. The ACOCD will become involved if there are signs that the death might have been occasioned by asbestosis; alternatively, what could be asked of the ACOCD is to determine whether the individual might have become 100% impaired between the date of his last clinical examination and the time of his death. Where other causes of death are suspected, Drs. Ritchie and Gray, rather than the ACOCD, will be consulted. The pattern of consultations actually followed in concrete instances is not necessarily as tidy as this description implies, but this befits a situation where, in point of fact, the information at hand is often spotty, contradictory, or both. As Professor Barth observed, there is frequently a disparity in the stated cause of death as between the death certificate and the Registrar General's form; death certificates often contain inadequate or incorrect information; and there has been a declining probability of autopsy in Ontario.[107] For his part, Dr. McCracken pointed out in testimony that the utility of hospital records varies with the care with which clinicians write their notes, and there are clinicians whose ". . . notes leave much to be desired. . . ."[108]

As best he can under the circumstances, the Board physician must find whether or not a claim warrants the payment of survivor benefits under the terms of the Act. He reports his finding to the Claims Adjudication Branch which, as usual, will grant the benefits if the recommendation is positive and forward the file to the Claims Review Branch if the recommendation is negative. The Claims Review Branch, in turn, does not appear to deviate from its normal practice of accepting the Board physician's finding without question and notifies the survivor, usually the widow, that the Board is unable to grant entitlement to dependency benefits.

At this juncture, what seems to differentiate the survivor benefit claims of deceased asbestotics from other asbestos-related claims is an enhanced likelihood that appeals will be launched. The non-existence of

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[106] Barth, *Workers' Compensation and Asbestos in Ontario*, p. 3.8.

[107] Ibid., p. 3.7.

[108] RCA Transcript, Evidence of Dr. William J. McCracken, 20 July 1982, Volume no. 53, p. 22.

Board statistics on appeals precludes quantification, but this impression is sustained by the testimony of the Executive Director of the Board's Claims Services Division[109] and borne out (perhaps magnified) by the media publicity that some of these appeals have attracted.

That claim denials in cases of deceased asbestotics should be likely to give rise to appeals is readily understandable. The stakes — income support for a widow who likely is without other significant resources — are high. And the contentious nature of the situation is acutely accentuated by the fact that the deceased — precisely because he was in receipt of a permanent partial disability pension — was suffering from a disease whose direct work-relatedness was recognized by the Board.

Many of the adverse client perceptions that have so tainted the Board's image are associated with claim denials and appeals in cases of deceased asbestotics. We have not acquainted ourselves, save for one exception noted below, with the details of individual claims. But Professor Barth, who reviewed a sample of claims on our behalf, observed that the concept of aggravation rarely seems to enter into the consideration of such claims.[110] Dr. Annalee Yassi, who reviewed another sample of claims for the Weiler inquiry, reported many instances of conflicting medical opinion, even among Board doctors themselves. She noted, with respect to at least one-quarter of the claims which she reviewed, that it was not obvious to her why the claim was either allowed or rejected.[111] Then there is the celebrated case of the late Mr. John Dodds, whose explicit details were made part of the record of our own inquiry by his widow, Mrs. Odette Dodds.[112] This case offers one of the rare instances in which the Corporate Board itself saw fit to make the ultimate appeal decision. The Corporate Board allowed this claim on the ground that surgery on a thyroid condition had not been performed, on the advice of the claimant's doctors, due to the claimant's asbestosis. As this claim progressed through the appeals structure, it is evident that the Board's doctors supported its denial on grounds that led them to go beyond disputable medical evidence to question the competence of the treating physicians.

Everything we have said in endorsing a revised Board appeals structure, with its favourable implications for the balancing of different medical opinions, applies with force to claims of deceased asbestotics. Presently, our attention must focus upon the level of primary adjudication. Here, what we distil from the observations of researchers and the record of our inquiry disturbs us. Our paramount impression is that Board adjudicators do not appear, on balance, to have approached the claims of deceased asbestotics with a mind-set attuned to the reality of medical uncertainty. This contrasts with the impression we formulated in the realm of asbestos-related lung cancer claims that fall short of satisfying the relevant Board guideline. In this realm, on the basis of the evidence cited in Chapter 12, we expressed the opinion that Board adjudicators give no appearance of a negative mind-set. Why should two realms of primary adjudication involving basically the same Board personnel create these contrasting impressions? We do not pretend to be able to answer this question in any definitive way, but we are instinctively drawn to attach importance to the obvious: in the realm of asbestos-related lung cancer, adjudication is guided by a Board eligibility rule; in the realm of deceased asbestotics, it is not.

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[109] RCA Transcript, Evidence of Mr. John F. McDonald, 13 July 1982, Volume no. 48, pp. 12–13.

[110] Barth, *Workers' Compensation and Asbestos in Ontario*, pp. 3.8–3.9.

[111] Yassi, "Occupational Disease and Workers' Compensation in Ontario: A Report for the Weiler Inquiry," chap. 4.

[112] Odette Dodds, Written submission to the Royal Commission on Asbestos, #75, 28 September 1981; and RCA Transcript, Submission by Mrs. Odette Dodds, Asbestos Victims of Ontario, 5 July 1982, Volume no. 47(B), pp. 12–20.

C.2 Structuring the Board's Discretion

We need hardly review the general arguments, developed at length in Chapter 12, for structuring the Board's discretion through formal eligibility rules whose content is known to both adjudicators and claimants. Such rules serve the goal of horizontal equity and simplify adjudication. In the process, they permit primary adjudicators to focus their attention on the claims that deserve it most: namely, the claims which, because they do not satisfy all the terms of the rules, warrant detailed scrutiny. We can find no reason to countenance the continued absence of an eligibility rule in the realm of deceased asbestotics. The Board has dealt with a substantial number of claims in this realm; such claims are therefore anything but unfamiliar.

The matter of devising an eligibility rule is facilitated by the approach that we have recommended be followed in determining the percentage impairment of asbestosis victims. Once due allowance is made for psychological as well as physical impairment, individuals whose asbestosis has been clinically measured as Severe will be deemed 100% impaired. Upon death, this 100% impairment guarantees survivor benefits pursuant to section 43(7) of the Act, whose basic intent is carried forward by the legislative exposure draft in the *White Paper on the Workers' Compensation Act.*[113] Accordingly, the matter of devising a specific eligibility rule for deceased asbestosis victims is confined to those who suffered from Mild or Moderate impairment, and we would not have it otherwise. We deem the automatic survivor benefits that flow from section 43(7) particularly appropriate to the situation of victims whose death occurs in the more advanced stages of asbestosis. While there is always room for medical uncertainty, the possibilities that the death of an asbestotic was not related to his condition are distinctly narrowed above the Mild and Moderate categories of impairment. For example, bronchial pneumonia is a common terminal illness, whether or not an individual is suffering from asbestosis. However, asbestosis makes individuals more vulnerable to bronchial pneumonia, and we have already cited testimony on the relationship between bronchial pneumonia and severe asbestosis.[114] Again, in the case of coronary artery disease, there is little direct evidence of any relationship between asbestosis and the development of coronary artery atherosclerosis and thrombosis. But if asbestosis is severe enough to diminish the oxygen level in the blood, inadequate oxygen supply to the cardiac tissues could contribute to the fatality of asbestotics also afflicted with coronary disease. With respect to the strength of the linkage between asbestosis and various immediate causes of death, individuals with Severe impairment are in our judgement appropriately distinguishable from those with Mild or Moderate impairment.

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[113] "Appendix I: Draft Bill to Amend the *Workmen's Compensation Act*, s. 30(2)," in Ontario, Ministry of Labour, *White Paper on the Workers' Compensation Act* [Toronto: Ontario Ministry of Labour, 1981], p. 26.

In the matter of devising an eligibility rule for deceased asbestosis victims who suffered from Mild or Moderate impairment, we find it reasonable for the time being to derive the terms of the rule from current Board practice. According to the testimony already cited, survivor benefits are invariably allowed where the victim of asbestosis died of this disease, or of mesothelioma, lung, laryngeal, or gastrointestinal cancer, or *cor pulmonale*. While there is a certain redundancy, given already existing Board guidelines, in naming all these immediate causes of death in the eligibility rule, we deem this desirable to ensure that the deceased claimant's survivors are fully informed. This eligibility rule, like all other Board-promulgated industrial disease rules, should be reviewed from time to time by our proposed Advisory Council on Industrial Disease Policy. We recommend that:

13.9 The Workers' Compensation Board, by directive of the Corporate Board, should promulgate an eligibility rule whereby survivors of deceased individuals who were rated by the Board as suffering Mild or Moderate impairment from asbestosis shall be entitled to benefits if death resulted from asbestosis, mesothelioma, lung, laryngeal, or gastrointestinal cancer, or cor pulmonale.

The above eligibility rule will, of course, leave a number of claims for detailed scrutiny on a case-by-case basis. These cases are difficult. Individuals who were mildly or moderately impaired may die from a variety of causes that more or less arose from their asbestotic condition or that were aggravated because the presence of asbestosis narrowed the range of effective medical treatment. Given medical uncertainty, benefit of doubt has a central role in case-by-case adjudication. Of course, this is as true of disease claims as it is of survivor claims. Therefore, we presently address the importance of benefit of doubt in a general context.

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[114] RCA Transcript, Evidence of Dr. Cameron C. Gray, 16 July 1982, Volume no. 51, p. 52.

D. Benefit of Doubt Policy

The Board's benefit of doubt policy was described in Chapter 12 and has been referred to at a number of points in the present chapter. In our judgement, a policy of this kind is eminently desirable and justifiable on three grounds. First, benefit of doubt provides a fair and practical way of addressing the medical uncertainty that enshrouds a substantial volume of industrial disease claims. Second, benefit of doubt combats what, in any agency endowed with broad administrative discretion, is the danger — particularly acute when guidelines or eligibility rules are in place — that adjudicators might approach claims which do not satisfy the terms of the rule with a negative mind-set. Third, benefit of doubt is fully in line with the historic tradeoff whereby workers' compensation was provided in lieu of the common law right of employees to sue their employers for damages. In civil liability cases, the plaintiff must prove his case on the balance of probabilities. This means that if the evidence is evenly balanced as between the plaintiff and the defendant, the plaintiff will not succeed. Benefit of doubt is consistent with the thrust of holding workers' compensation claimants to standards that enhance their chances of recovery from the chances they would have if their common law rights had not been taken away.

The Board's benefit of doubt policy serves all of the above. Its application, however, is uneven. In line with benefit of doubt is our finding that asbestos-related lung cancer claims outside the terms of the relevant guideline have not been approached with a negative mind-set. At odds with benefit of doubt, on the other hand, is our finding that the adjudication of claims involving deceased asbestotics has not been attuned to medical uncertainty. Meantime, with respect to assessing the degree of impairment occasioned by asbestosis, the benefit of doubt policy is not communicated to the ACOCD. The result, because ACOCD findings are accepted in turn by the Board physician and by non-medical adjudicators, is that benefit of doubt is mute in the realm of asbestosis.

There is an evident need to ensure the even application of benefit of doubt in Board adjudication. This can be promoted by elevating benefit of doubt to the level of a statutory rule. The legislative exposure draft that accompanies the *White Paper on the Workers' Compensation Act* contains an important clause to this effect. Section 6(7) of the exposure draft states the following:

> In determining any claims for benefits under this Act the decision shall be made in accordance with the merits and justice of

> the case and where there is doubt on an issue and the disputed possibilities are evenly balanced, the issue shall be resolved in accordance with that possibility which is favourable to the worker.

That this language gives statutory effect to benefit of doubt is entirely apparent. In civil liability cases, as we have pointed out, the plaintiff will not succeed if the balance of probabilities is even. Section 6(7) stipulates that an even balance shall be resolved in favour of the claimant. We are in accord with elevating benefit of doubt to the level of the *Workers' Compensation Act*.

To be sure, the language of the legislative exposure draft is not the only way to give statutory expression to benefit of doubt. A particular example of alternative language was brought to our attention at our final public hearing by counsel for the Asbestos Victims of Ontario and the Iron Workers' Union, Mr. David Starkman.[115] This example is found in section 85 of the *Pension Act* of the Parliament of Canada.[116] In the matter of pensions for past or present members of the armed forces who have been disabled or died as a result of military service, the Canadian Pension Commission and its emanations are instructed, in section 85, to apply benefit of doubt in the following words:

> 85. The Commission, an Entitlement Board and the Pension Review Board shall, in determining the entitlement of an applicant to an award and in assessing the extent of the disability of a member of the forces to or in respect of whom entitlement to a pension has been established
>
> (a) draw from all the circumstances of the case and all the evidence presented to it every reasonable inference in favour of that applicant or member, and
>
> (b) accept as proof of any fact that the applicant or member is required to prove, any credible evidence submitted by him that is not contradicted and where, in weighing any evidence submitted to it, any doubt exists as to whether the applicant or member has established his case, the Commission, an Entitlement Board or Pension Review Board, as the case may be, shall resolve such doubt in favour of the applicant.

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[115] RCA Transcript, Submission by Mr. David Starkman on behalf of the Asbestos Victims of Ontario and the International Association of Bridge, Structural and Ornamental Iron Workers, Local 721, 28 January 1983, Volume no. 58, pp. 19–20.

[116] *Pension Act*, R.S.C. 1970, c. P-7, as am. by R.S.C. 1970 (2d Supp.), c. 22, s. 85.

As we read it, the degree of benefit of doubt spelled out by the *Pension Act* is at least as great as that articulated by the language of the *White Paper* legislative exposure draft. It is arguable that the degree of benefit of doubt accorded by the federal statute might be somewhat greater; were this to be so, our respect for the elusive boundaries of medical uncertainty would lead us to favour its formulation. Whatever the case, a decisive consideration in favour of the language of the federal *Pension Act* lies in what we discern as its fuller elaboration of benefit of doubt and consequent clarity to persons who are not legally trained. The latter characteristic, of course, describes the Board's physicians, its lay adjudicators, and the vast run of its claimants. Concluding as we do that benefit of doubt should be elevated to the level of the *Workers' Compensation Act*, and that the language of the federal *Pension Act* provides an attractive model on which to base the pertinent clause, we recommend that:

13.10* *The Workers' Compensation Act should be amended so as to extend to claimants benefit of doubt in a manner consistent with that found in the Pension Act of the Parliament of Canada.

E. Communicating with Claimants

Whether statutorily enshrined or promulgated by the Corporate Board, eligibility rules, benefit of doubt policy, and review or appeal mechanisms are instruments meant to ensure that justice is done to workers' compensation claimants. But justice must also be *seen* to be done. What is by now a tiresomely reoccurring theme in our review of Board practice with respect to asbestos-related disease is that the Board has too often veiled otherwise commendable efforts to promote justice from the view of its claimants.

Much more can and should be done to dispel the aura of mystery that has so promoted the perception of the Board as arbitrary and capricious. Our recommendations concerning the public dissemination of eligibility rules and the importance of written appeal decisions are measures designed to serve this end. But however publicly it may be disseminated, the quantity and complexity of information concerning Board practices ensures that it will only be grasped fully by a knowledgeable few. We therefore consider it essential that the Board adopt a policy of communicating pointed information, rationed on the basis of its pertinence, directly to the individual claimants to whom it communicates its decisions.

We have sampled a number of the letters through which the Board informs claimants of the disposition of their cases. Four examples of the letters that are sent to asbestosis claimants were reproduced in Professor

Barth's study.[117] We also secured from the Board examples of the letters that are sent to survivors of deceased asbestotics.[118]

The letters reproduced by Professor Barth included: (i) a letter denying a claim for a partial disability pension; (ii) a letter granting a claim for a partial disability pension; (iii) a letter reporting that given a finding of no progression in an asbestotic condition, no change in the amount of partial disability pension is warranted; and (iv) a letter reporting that given the recognition of a higher degree of impairment at the most recent clinical examination, the partial disability pension is being increased. All four letters contain the following identical paragraph:

> If you have any reasons for objecting to this decision or have any concerns or questions about the matter, please let us know as soon as possible.

The appearance of this paragraph in all four letters is warranted because, in the case of a progressive disease like asbestosis, even a successful claimant might believe that there are grounds on which to appeal for a higher partial disability pension. However, the wording of this paragraph makes no explicit mention of the claimant's *right* to appeal; even less does it tell the claimant how to go about launching an appeal. The Board publishes a useful booklet, in five languages, on how to appeal its decisions.[119] This booklet, among other things, refers to the services of the Workers' Advisors, whose services are available without charge to aid in preparing and presenting appeals. We have no indication that this booklet is enclosed with the letters just cited as a matter of course.

The letters sent to survivors of deceased asbestotics differed from the four sent to asbestosis victims. The two such letters we sampled, both reporting denial of survivor benefits, state explicitly:

> The above decision is open to appeal and information on the appeals procedure may be found in the attached pamphlet.

This is self-evidently a much clearer communication. We urge that the Board standardize its communications with claimants so as to ensure, in all appropriate instances, that the claimant understands that he has a right to appeal and possesses the publication on how to launch an appeal. Still more information is desirable. Whenever a disease claim falls in a realm that is encompassed by an eligibility rule and is denied by the Board as failing to satisfy the terms of that rule, the claimant should be sent a copy of the rule in question. We recommend that:

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[117] Barth, *Workers' Compensation and Asbestos in Ontario*, pp. 2.37–2.40.

[118] Letter and attachments from Mr. Alex Joma, Secretary, Workmen's Compensation Board to the Royal Commission on Asbestos, 3 August 1982.

[119] Letter and attachments from Mr. Alex Joma, Secretary, Workmen's Compensation Board to the Royal Commission on Asbestos, 23 August 1982, including WCB booklet, *Making an Appeal.*

13.11 The Board should adopt a standardized form of communication with claimants which ensures:

(i) that all claimants for whom it is pertinent shall know that they have an explicit right to appeal and are informed of the procedures for launching an appeal; and
(ii) that all claimants whose disease is the subject of an eligibility rule shall have a copy of the pertinent eligibility rule.

With respect to the survivors of deceased asbestotics, there is a special matter to which Professor Barth called attention. This is that the deceased claimant's survivors may not be aware that the Board is proceeding to adjudicate a question of great economic importance to them.[120] As we have pointed out, the death of an individual who was in receipt of a disability pension does not occasion a new claim for survivor benefits; the Board quite properly considers whether such benefits are warranted as a matter of course. But it is only claims arising from the death of an individual already rated as 100% impaired which can, by virtue of section 43(7), be adjudicated expeditiously. In all other instances, either the cause of death must be determined or the ACOCD must be asked whether the claimant might have become 100% impaired since the date of his last examination. The time required for the adjudicative process may well be drawn out by the sketchy information in the hands of the Board, information which the survivors might be in a position to supplement.

In our judgement, the situation can be ameliorated through two measures. First, individuals who are in receipt of partial disability/impairment awards should be informed of the rules pursuant to which their survivors may be eligible for benefits in the event of death. Second, in all instances where the Board has been unable to grant survivor benefits within one month of death, the claimant's survivors should be informed that the Board is adjudicating the matter, given copies of the pertinent eligibility rules, and asked to bring forward any relevant information. We recommend that:

13.12 The Workers' Compensation Board should adopt a standardized practice whereby:

(i) recipients of partial disability/impairment awards are informed of the rules pursuant to which their survivors may be eligible for benefits in the event of death; and

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[120] Barth, *Workers' Compensation and Asbestos in Ontario*, pp. 3.11, 9.12-9.13.

(ii) in all instances where the Board has been unable to grant survivor benefits within one month of death, the claimant's survivors shall be notified that the matter is under adjudication, provided with the pertinent eligibility rules, and asked to bring forward any relevant information.

Appendix to Chapter 13
Ontario Workers' Compensation Board
The Adjudication of Claims for Psychotraumatic Disability

An employee is entitled to benefits when personal injury is sustained through an accident which arises out of and occurs in the course of employment. "Injury" includes both physical and emotional disability.

1. General Rule

Where it is evident that a diagnosis of a psychotraumatic disability is attributable to a compensable injury or its sequelae, entitlement shall be granted providing that the psychotraumatic disability became manifest within five years of the injury, or within five years of the last surgical procedure.

Psychotraumatic disability is considered to be a temporary condition. Only in exceptional circumstances will this type of disability be accepted as a permanent condition.

2. Psychotraumatic Disability Entitlement

Entitlement for psychotraumatic disability may be established when the following circumstances exist or develop:

2.1 Organic brain syndrome secondary to: traumatic head injury, toxic chemicals including gases, hypoxic conditions or conditions related to decompression sickness.

2.2 As an indirect result of a physical injury.

2.2.1 Emotional reaction to the accident or injury.

2.2.2 Severe physical disability.

2.2.3 Reaction to the treatment process.

2.3 The psychotraumatic disability is shown to be related to extended disablement and to non-medical, socio-economic factors, the majority of which can be directly and clearly related to the compensable injury.

3. Other Factors

The following relevant points should be evaluated in assessing the extent of psychotraumatic disability entitlement:

3.1 *Prior History* — In all cases where history of a prior psychiatric condition has been shown to exist, the question of allowance on an aggravation basis shall be considered, having regard for the emotional effect of the occupational occurrence and its sequelae.

3.2 *Unrelated Psychiatric Disability* — In some cases, psychiatric disability may become apparent in an otherwise uneventful case, and enquiry establishes its origins to other factors (such as family crisis), having no relationship whatsoever to the accident. On medical advice and under the provisions of section 23 of the Act, allowance may be made for concurrent treatment. The psychiatric treatment will be in order during the period of active treatment for the physical disability, so long as such treatment enhances the recovery of the physical disability, in the opinion of the Board.

4. Second Injury and Enhancement Fund Relief

In claims for psychotraumatic disability, the Board approved policy regarding the aggravation of pre-existing conditions and application of the Second Injury and Enhancement Fund shall be applied.

5. Benefit of Doubt

Claims which do not meet the above criteria shall be individually judged on their own merits having regard for the evidence submitted. The benefit of doubt applies.

Approved by the Board February 9, 1982.

SOURCE: The Workmen's Compensation Board, Minute #11, 9 February 1982, p. 4951.

Chapter 14 Rehabilitation, Outreach, and Prevention

A. Rehabilitation: Retrospect and Prospect

The Workers' Compensation Board has made pioneering efforts in the matter of rehabilitating employees whose past exposure to a hazardous substance may constitute cause for removing them from further exposure. Specifically in the realm of asbestos, the Board devised a Special Rehabilitation Assistance Programme (SRAP) whose primary focus was upon the employees of the Johns-Manville plant in Scarborough. In support of this Programme, Board physicians devised a rule which to the best of our knowledge is unique to Ontario: the Asbestos Fibre Dust Effect (AFDE) guideline.

The SRAP experience has been hedged by controversy, both within the Board and externally. More importantly, the SRAP experience has portents for the future. This is because the Regulation Respecting Asbestos formulated by the Ministry of Labour features explicit provisions covering the removal of designated workers who have been exposed to asbestos and assigns a specific role to the Workers' Compensation Board in their rehabilitation. We shall first examine the SRAP experience and then assess the prospects of the approach envisaged by the Asbestos Regulation.

A.1 The Special Rehabilitation Assistance Programme

By 1975, the outline of what this Report has categorized as a world-class occupational health disaster at the Johns-Manville plant in Scarborough had become sufficiently discernible to preoccupy the Ontario Legislature. Political pressure from organized labour, coupled with concerns expressed by members of the New Democratic Party on the floor of the

Legislative Assembly,[1] prompted the Workers' Compensation Board to devise a special programme for asbestos workers. The Board's efforts in this regard were initiated in October 1975.[2]

The Board launched these efforts from a base of experience in worker rehabilitation which has earned it a reputation such that, in Professor Peter S. Barth's words, ". . . Ontario is held up around the world as a model jurisdiction in the area of rehabilitation."[3] In the realm of industrial disease occasioned by exposure to hazardous dust, the Board, earlier in 1975, had taken an unprecedented initiative announced in May by the Minister of Labour. This initiative targeted upon workers who had been adversely affected by exposure to silica dust, radiation, or both in the Elliot Lake uranium mines.[4] In fashioning a special programme tailored to asbestos workers, the Board had reason to be conscious of its Elliot Lake initiative.

The Board assigned the task of devising a programme for asbestos workers to its Claims Services Division and to what was then its Rehabilitation Services Division.[5] The latter included all the organizational elements which subsequently, in 1978, were designated as the Medical Services Division.[6] In performing their task, the Board physicians, including the Board's Chest Disease Consultant, Dr. Charles Stewart, faced a challenge: to find a means of identifying workers who might be eligible for rehabilitation yet who had not already been afflicted by asbestos-related disease. This, of course, was crucial to the very concept of a special rehabilitation programme: individuals already suffering from asbestosis were eligible for the Board's rehabilitation services as a matter of course; the central idea of a special programme was to extend rehabilitation to persons not already suffering from asbestosis.[7]

In the case of Elliot Lake miners exposed to silica dust, this question had been easily resolved. The Elliot Lake situation called for the determination of a medical condition that could be taken as a harbinger of silicosis: a state of pre-silicosis. The material for making this determination was readily at hand.[8] Chest stations for the surveillance of miners had been established throughout Ontario in 1929. The activity of these stations wrought the development of an x-ray classification scheme based on a South African system. The scheme featured the following codes: Code One was a perfectly normal chest; Code Two involved a slight increase in markings; Code Three was a moderate increase in linear markings; and Code Four denoted a beginning of lace-like or net-like change called arborization. In Dr. Stewart's words, the latter involved ". . . some obscuring of the normal pattern, possibly also including some very small pinpoint, pinhead-size inclusions that were not recognized as nodules."[9] The next stage — Code Five — revealed frank nodulation and hence permitted a diagnosis of silicosis. Accordingly, Code Four could be taken as identifying pre-silicosis. The confidence with which this conclusion could be drawn was supported by what over the years had developed into a statistical system embracing 60,000 miners with hundreds of thousands of associated records.[10] Code Four was therefore designated as the threshold for entry into the Board's Elliot Lake removal and rehabilitation programme; this later received the approbation of the Royal Commission on the Health and Safety of Workers in Mines (the Ham Commission), which was sitting at the time this programme was devised.[11]

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[1] Energy and Chemical Workers Union, Written submission to the Royal Commission on Asbestos, #32, January 1980, pp. 24–27.

[2] The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, p. 56.

[3] Ontario, Royal Commission on Asbestos, Transcript of Public Hearings [hereafter RCA Transcript], Evidence of Professor Peter S. Barth, 24 August 1982, Volume no. 57, p. 104.

[4] The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, p. 53.

[5] Ibid., p. 56.

[6] RCA Transcript, Evidence of Dr. William J. McCracken, 20 July 1982, Volume no. 53, pp. 5, 92.

[7] RCA Transcript, Evidence of Mr. John F. McDonald, 13 July 1982, Volume no. 48, pp. 143–145.

In fashioning a special programme for asbestos workers, the Board physicians had to find a means of identifying individuals whose condition could be taken as a harbinger of asbestosis. Chest surveillance records of asbestos workers went back to 1947, but statistics had not been developed.[12] Furthermore, the x-ray code that permitted the discernment of pre-silicosis could not identify pre-asbestosis. In the asbestos case, according to Dr. Stewart's testimony, x-ray changes differ because ". . . the changes in pre-asbestosis are in the lower lung fields and they are interstitial and linear, they are not nodular."[13] Given the consequent irrelevance of Code Four to asbestos-exposed workers, the Board physicians had to examine individual x-ray reports from the Chest Surveillance Programme. These reports, rather than coding changes beyond Code Three, would describe them. They would describe, in Dr. Stewart's words, ". . . changes that relate to the linear pattern in the lower lung field as being slightly increased, or slight interstitial change suggestive of mild fibrosis, slight blunting of the costophrenic angles."[14] On the basis of such descriptions, the Board physicians fashioned what was approved by the Corporate Board as the Asbestos Fibre Dust Effect (AFDE) guideline. The text of this guideline is as follows:

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[8] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, pp. 14–24.

[9] Ibid., p. 16.

[10] Ibid., p. 18.

[11] Ontario, *Report of the Royal Commission on the Health and Safety of Workers in Mines* (Ham Report), James M. Ham, Commissioner (Toronto: Ministry of the Attorney General, 1976), pp. 56–57.

[12] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, pp. 106–107.

[13] Ibid., p. 23.

> It is recommended that a case be accepted as showing AFDE when the following circumstances apply:
>
> 1. — an adequate history of exposure is documented — a minimum of 10 years unless an unusual intensity exposure is established.
>
> 2. — when at least two of the following radiological signs are present:
>   2.1 — intralobar pleural thickening (major or minor fissures);
>   2.2 — variable obliteration of the costophrenic angles;
>   2.3 — variable pleural thickening of a focal or plaque-like nature along the lateral chest wall or diaphragm is noted.
>
> 3. — and when in addition at least two of the further following radiological signs are present:
>   3.1 — horizontal linear markings 1.3 mm. thickness in the lower zones usually bilateral;
>   3.2 — a general coarsening of the lower zone linear pattern with partial replacement by a reticular or net-like pattern;
>   3.3 — superimposed of minute bead-like opacities, 1-2 mm. in diameter over or adjacent to the lower zone pulmonary arterial tree.
>
> 4. — these changes described above must have occurred during the preceding 5- to 10-year period.
>
> 5. — the vital capacity studies may have shown changes over the past 5 years although the last result is still within normal limits.[15]

With the AFDE guideline in place, the Special Rehabilitation Assistance Programme was announced amidst considerable publicity by the Minister of Labour on May 13, 1976. In line with the Elliot Lake programme, the SRAP extended Board assistance, on a voluntary basis, to employees who, either because they had asbestosis or met the terms of the

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[14] Ibid., p. 22.

[15] Workers' Compensation Board, Claims Services Division, *Board Policies and Administrative Directives* [manual continually amended], pp. 197C-197D.

AFDE guideline, wished to be removed from the risk of further exposure to asbestos. Assistance could take the form of Board rehabilitation benefits designed to make up the difference between former wages and lower wages in non-exposure employment. Alternatively, benefits up to the equivalent of full compensation were available if the employee wished to undergo Board-financed retraining for suitable non-exposure employment and could be applied to make up any wage difference in employment that followed retraining. Benefits were also payable, along with travel and removal allowances, to employees who might seek or receive work in new locations. The administration of the SRAP came under the Board's Vocational Rehabilitation Division. Consideration for admission to the SRAP involved an initial employee interview with a team composed of representatives of this Division, the Medical Branch, and the Claims Adjudication Branch.[16]

Interviews were initiated within a month of the ministerial announcement of the SRAP. It was clear from the Programme announcement that an individual must be either asbestotic or satisfy the AFDE guideline to be eligible. But a crucial programme element was missing and yet to be determined. This was the question of designating the employment from whose associated exposure risk a worker might seek to be removed.

In the case of the Elliot Lake programme, the straightforward answer had been to designate underground work as risk employment, thereby extending rehabilitation benefits to miners who wished to remove themselves from exposure risk either by working above ground or by seeking work in other mines. In this connection, it is to be noted that the Elliot Lake programme aimed to reduce *relative* exposure risk. As Dr. Stewart testified, ". . . one of the underlying planks of the [Elliot Lake] programme in its conception was the acceptance . . . of low exposure alternatives . . . . So that there was never, from the start, the necessity that there be an absolute lack of exposure in the alternative job location. . . ."[17]

Even as the SRAP interviews proceeded at the Johns-Manville plant, the Board physicians had to unravel the outstanding question of what constituted risk employment in that plant. This question was not resolved until January of 1977. The Board awaited while a new set of dust samples was taken by the Occupational Health Branch of the Ministry of Labour. On the basis of the sample results, the Board's Chest Disease Consultant, Dr. Stewart, recommended that the Transite pipe production portion of the plant be designated as its only hazardous area.[18] Transite pipe shipping and receiving should not be designated because some of the work was done out of doors and the range of fibre counts inside was ". . . mostly under 0.5

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[16] Ibid., pp. 197E–197G.

[17] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, pp. 106–107.

[18] Ibid., p. 104.

fibres."[19] The Corporate Board accepted Dr. Stewart's recommendation. Accordingly, the only employees eligible for the SRAP would be those who, being asbestotic or meeting the AFDE guideline, worked in Transite pipe production or staffed the plant's erection and repair division. The latter were included because ". . . a great portion of their time and duty was spent in the Transite pipe production area on maintenance repair work. . . ."[20]

All this was delineated in January 1977. In a setting where interviews had been ongoing for seven months, grounds for confusion could scarcely have been more fertile. It becomes entirely understandable to us why workers, or for that matter union officials, might have formed the impression that asbestosis or AFDE alone constituted the necessary and sufficient condition for entry into the SRAP. A programme which, at its inception, spoke of removal from risk employment without designating where such employment might be located invited the interpretation that the risk area might well encompass the entire plant. Board officials themselves were caught up in the confusion. During cross-examination by Mr. Ed Cauchi of the Asbestos Victims of Ontario, the Board's Rehabilitation Specialist, Mr. William D. Pearce, testified that between June 1976 and January 1977, individuals admitted into the SRAP included employees from the shipping department, from fibreglass and Thermobestos, and even one salaried employee.[21] These individuals were retained in the Programme after January 1977, in deference to the Board's benefit of doubt policy.[22] This, however, only added the perception of inequity to the indignation of employees subsequently denied entrance to the Programme because they were not in the designated risk area. Sentiments of confusion, frustration, and inequity still ran high in the summer of 1982, when we heard formal testimony on the SRAP.

And with the passage of time, the SRAP had not stood still. Eligibility criteria were redefined when Transite pipe manufacturing was terminated at Johns-Manville in May of 1980. Following the shutdown, and in the wake of consultations between the Minister of Labour and the Board, it was announced that, with respect to ex-employees in Transite pipe who were employed elsewhere in the plant, the Board would waive the requirement for being at risk if it appeared to the Board that for medical reasons such employees appeared incapable of carrying out their new duties.[23] In the result, the SRAP was no longer a programme to remove workers with asbestosis or AFDE from further exposure to asbestos. It was instead what could be termed a pilot programme in compensating and rehabilitating workers who were medically impaired, or might become impaired, on the basis of their socio-economic disability. Other changes that had been made were in line with this thrust: the Board had decided in 1977 that partial disability pensions for asbestosis were not to be deducted from wage-loss benefits; these benefits in turn had become open-ended when the Board removed an initial one-year limitation on their payment; and for its part, Johns-Manville in December of 1979 volunteered to continue its benefit coverage of workers entering the SRAP.[24]

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[19] Ibid., p. 105.

[20] RCA Transcript, Evidence of Mr. William D. Pearce, 19 July 1982, Volume no. 52, p. 8.

[21] RCA Transcript, Evidence of Mr. William D. Pearce, 12 August 1982, Volume no. 56, pp. 50, 52.

[22] Ibid., p. 51.

[23] Ibid., pp. 20–21.

The Board reported to us that, as of August 10, 1982, 83 individuals had been recognized over the life of the SRAP as being afflicted with asbestosis or AFDE.[25] Of these, 35 failed to qualify for the Programme because they had not been considered at risk. Forty-one did qualify: this total included the employees not at risk but deemed qualified in the confusion of the SRAP's early months, and also included 9 employees who qualified after the Board had waived the requirement for being at risk. Twelve of the 41 refused the Programme, leaving 29 who were admitted. Twenty-six individuals remained on the Programme as of August 10, 1982: 11 were employed at reduced wages and hence were receiving benefits to restore their earning power to its previous level; 6 were in formal training; 5 were being assessed for training or job placement; and 4 were undergoing medical treatment. Three individuals had left the Programme: 2 for retirement and one who was deemed fully rehabilitated, having found full-time employment in the field in which he had received the benefit of training.

What can be said of the SRAP experience? In his study for this Commission, Professor Peter S. Barth expressed the opinion that: "Rarely if ever has the WCB been involved with a less successful effort . . . . The programme could serve as the source for a lesson in public administration courses on how not to formulate and operate government programmes."[26] In its commentary on the Barth study, the Board made no reference to this severe judgement but considered ". . . that despite the conflicts, confrontations and difficulties [the SRAP] was a necessary venture from which some good may eventually result."[27] Our own observations are as follows.

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[24] Peter S. Barth, *Workers' Compensation and Asbestos in Ontario*, Royal Commission on Asbestos Study Series, no. 2 (Toronto: Royal Commission on Asbestos, 1982), p. 8.7.

[25] The data in this paragraph are drawn from Ontario, Royal Commission on Asbestos, Exhibit IV-8 [hereafter RCA Exhibit], in RCA Transcript, Evidence of Mr. William D. Pearce, 12 August 1982, Volume no. 56, p. 19: The Workmen's Compensation Board, Ontario, Data re Special Rehabilitation Assistance Programme.

[26] Barth, *Workers' Compensation and Asbestos in Ontario*, p. 8.1.

[27] The Workmen's Compensation Board, "Comments on the Study and Review Papers on Workmen's Compensation and Asbestos in Ontario," Toronto, 6 August 1982, p. 17. (Mimeographed.)

The SRAP had its genesis in the discernment of what was later documented as a world-class occupational health disaster. This did not permit the time required for a structured, integrated response. It is understandable that the pressure of time would barely permit the development of the new concept bound up in the AFDE guideline. At this point the confusion attendant upon launching a risk-removal rehabilitation programme before risk areas had been delineated poisoned the atmosphere in which the programme would operate. It then turned out that the SRAP no longer dealt with removal from risk after May 1980. Thereupon, if it is viewed purely as a rehabilitation programme, the SRAP can hardly lay claim to success if it is judged by the outcome of a single fully rehabilitated and active member of the labour force. On the other hand, the SRAP did have the humane impact of compensating socio-economic disability incurred by impaired or potentially impaired workers. In this connection, the Board's Chest Disease Consultant, Dr. Stewart, made a telling observation when he testified that the mean average age of the SRAP clients was ten years older than at Elliot Lake.[28] The self-evidently reduced potential of older workers for rehabilitation enhanced the importance of the SRAP's compensation element.

Professor Barth documented that the Board harboured honest reservations at the time the SRAP was being devised. He cited an internal Board memorandum which notes a widely acknowledged conundrum, namely, that ". . . it is by no means agreed that withdrawal from exposure will stop or slow progression of asbestosis."[29] Did the SRAP succeed in altering the medical condition of its clients? There is likely no basis for telling, given especially the confusion over who was and who was not removed from risk. The Board has confirmed to us that an unspecified number of asbestotics admitted to the SRAP did progress from their initial 10% disability rating, testifying to the intractable nature of this disease. More poignantly yet, the shadows of the disaster that generated the SRAP continue to lengthen: of the 83 individuals recognized for SRAP purposes as asbestotic or AFDE, 14 have died, 9 of them of causes deemed by the Board to be asbestos-related.[30]

Whatever else may be said about the SRAP, it offers anything but a firm base from which to assess the advisability of removal-rehabilitation programmes. On the other hand, certain elements of the SRAP, notably the AFDE guideline, may be a relevant legacy. Let us probe this in the context of the removal and rehabilitation features of the Regulation Respecting Asbestos.

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[28] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, p. 106.

[29] Barth, *Workers' Compensation and Asbestos in Ontario*, p. 8.4.

[30] Letter from Mr. Andy Emmink, Assistant Secretary, Workers' Compensation Board to the Royal Commission on Asbestos, 7 March 1983.

A.2 The Regulation Respecting Asbestos

The provisions of the Regulation Respecting Asbestos which envisage the possible removal and rehabilitation of workers who have been exposed to asbestos are found in the Code for Medical Surveillance, which is appended to the Regulation, and in section 16 of the Regulation. The relevant provision in the Code states:

> If the examining physician determines that signs of asbestos-induced disease are present the worker shall not be removed from asbestos exposure before consultation with the senior chest disease consultant of the Ministry of Labour and a rehabilitation officer of the Workmen's Compensation Board (WCB). To qualify for compensation or rehabilitation further assessment by the WCB will be necessary. If asbestos-induced disease is confirmed the physician shall then determine whether the worker is fit, fit with limitations or unfit and take action in accordance with Section 16 of the Asbestos Regulation.[31]

Before addressing section 16, we take due note that the above provision appears in the Code in response to concerns expressed to the Ministry of Labour by the Workers' Compensation Board. Dr. Stewart testified before us that an initial draft of the Regulation seemingly gave the examining physician the right to remove the worker on the basis of his own assessment; the worker might then end up with no compensation if the Board disagreed with the physician's act. By involving the Board, the Code appears to cover this eventuality.[32]

At this juncture, the relevant provisions of section 16 state that:

> (1) [The examining physician] shall advise the employer, who shall act thereon, and the worker whether the worker is fit or because of a condition resulting from exposure to asbestos is fit with limitations or unfit, to work in an asbestos exposure . . . .
>
> (2) Where a worker is removed from exposure to asbestos because a physical examination or clinical test discloses that the worker *may have or has* a condition resulting from exposure to asbestos *and suffers a loss of earnings occasioned thereby,* the worker is entitled to *compensation for the loss* in the manner

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[31] Code for Medical Surveillance of Asbestos Exposed Workers, s. 4(3), in Regulation Respecting Asbestos, O. Reg. 570/82, made under the *Occupational Health and Safety Act,* R.S.O. 1980, c. 321.

[32] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, p. 176.

> and to the extent provided by the *Workmen's Compensation Act*. (Emphasis added.)[33]

We attach importance to the words we have emphasized because we can only interpret them as enshrining the basic thrust of the Special Rehabilitation Assistance Programme in the Regulation Respecting Asbestos. In particular, we note the following. First, the reference to a worker who *may have* or *has* an asbestos-related condition plainly envisages, in addition to asbestos-related disease, a definable condition of pre-asbestosis. Second, the references to compensation for loss of earnings occasioned by removal envisage with equal clarity a Board programme which, as distinct from any partial impairment/disability pension arising from asbestos-related disease, provides benefits similar to those of the SRAP for individuals removed from risk.

These points being duly noted, we feel called upon to make a number of observations. The Regulation applies to all asbestos-related establishments, not to a single plant. In this sense, it has another element in common with the SRAP in that this programme, at least in principle, was not intended exclusively for Johns-Manville workers.[34] The fact remains, however, that the SRAP was developed and applied solely in the context of the Johns-Manville situation. In particular, as Dr. Stewart testified before us, the AFDE guideline was devised from an examination of serial films of Johns-Manville employees. The guideline's x-ray description criteria, accordingly, ". . . cannot be interpreted as . . . representing asbestos changes that might occur in other industries . . . ."[35] It follows that the Regulation, envisaging as it does a condition of pre-asbestosis, will find in the AFDE guideline what is at best a weak reed to lean upon: the guideline is derived from x-ray films of workers exposed in the course of a particular asbestos-using industrial process.

There is next the matter of removing the worker from asbestos exposure. From what exposure and to where? With respect to what exposure, the Regulation is silent on the question of whether removal is to be to no occupational exposure or to a relatively lower occupational exposure. As was pointed out earlier in this chapter, both the Elliot Lake programme and the SRAP accepted removal to lower exposure alternatives. It may be that the Regulation wishes to leave the question of what exposure to the Workers' Compensation Board, whose consultation in the matter of removal is mandated by the Code. But there is also section 16(5) of the Regulation, pursuant to which the examining physician's finding that a worker ". . . is fit with limitations or is unfit for work in an asbestos exposure. . ." shall be communicated to the Chief Physician, Occupational Health Medical Service of the Ministry. More likely, then, the matter of what exposure is being left to joint determination by Ministry and Board officials. The fact remains that the Regulation is silent on a matter of policy that is ripe with controversy: whether a worker is to be moved to relatively lower exposure or to no exposure. Closely connected is the question of where the worker is to be removed. The late delineation of risk areas from non-risk areas in the Johns-Manville plant haunted the SRAP. A Regulation written for the entire asbestos-related industry can hardly be expected to pinpoint risk areas in plants, but a Regulation that is silent on the matter of designation invites future repetitions of the SRAP experience.

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[33] Regulation Respecting Asbestos, s. 16.

[34] RCA Transcript, Evidence of Dr. William J. McCracken, 20 July 1982, Volume no. 53, p. 119.

[35] RCA Transcript, Evidence of Dr. Charles Stewart, 15 July 1982, Volume no. 50, p. 121.

Is the removal of a worker who is "fit with limitations or unfit, to work in an asbestos exposure" voluntary or compulsory? On this crucial question, the Regulation is mute. For that matter, the Regulation does not tell us the relationship between being a worker who "may have or has a condition resulting from exposure to asbestos" and being "fit with limitations or unfit." The SRAP, to its credit, was a voluntary programme. As we have seen, 12 workers refused to enter it. Individual workers may have any of a number of entirely valid reasons for refusing removal and rehabilitation. Reflecting upon these, the Executive Director of the Board's Claims Services Division pointed out in testimony that employees whose seniority represents a major investment in company benefits may well be loath to consider the uncertainties of retraining and/or alternative employment, particularly when the possibility that removal has beneficial health effects is speculative.[36] It may be that the Regulation looks to the Board to protect the choice of workers. Nonetheless, we view the absence of an explicitly recognized right of workers to refuse removal and rehabilitiation as an omission that cannot be condoned. Altogether, to borrow the expression used by Professor Barth in another context, we consider that the sections of the Regulation Respecting Asbestos which deal with removal and rehabilitation could well serve as a lesson in public administration courses on how not to draft regulatory instruments.

What is to be done in the matter of removal and rehabilitation for asbestos workers? The essential starting point, in our view, lies in the health evidence we have presented earlier in this Report. Cumulative exposures which, for the longest-term employees in friction plants, are unlikely to exceed 25 fibres per cubic centimetre-years (f/cc-yrs), should make asbestosis an extinct disease. It follows that removal and rehabilitation are irrelevant to workers who have become employed since asbestos became a designated substance, at least if asbestosis is the disease whose control is envisaged by these steps. For such workers, the disease that continues to be a source of concern is cancer. But the multiple causes of this disease make removal for any purpose other than medical treatment questionable in the extreme.

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[36] RCA Transcript, Evidence of Mr. John F. McDonald, 13 July 1982, Volume no. 48, pp. 149–151.

The potential clientele for removal and rehabilitation programmes is therefore narrowed, in the case of asbestos, to those workers whose employment history takes them back to the high exposures of the 1960s and, to a lesser extent, the 1970s. It is this employee population which, unhappily, will be the source of the future cases of asbestosis in Ontario. For them, however, the question remains whether any beneficial medical results are to be expected of removal from further exposure. The exposure levels from which such workers would be removed, when measured in relation to their working life exposure dose, are trivial. Not least for this reason, we emphasize that it is essential to vest an explicit right to refuse removal in every asbestos-related worker. Nor should it be left to an examining physician, however named for purposes of surveillance, to advise the employer, or anyone else, that a worker is "fit with limitations or unfit, to work in an asbestos exposure." (See Chapter 8.) In our view, the duty of the examining physician should be simply to assess whether a worker "may have or has a condition resulting from exposure to asbestos" and to report thereon to the worker and to the Workers' Compensation Board.

We consider that the Board, upon receiving such an assessment, should thereupon set up a claim for the worker involved. Both to protect the employee, and to guard against the perception that examining physicians may be beholden to employers, we consider that the responsibility of informing employers should be vested in the Board. This will follow from the act of setting up a claim. More importantly, however, the setting up of a claim will permit, subject to all the safeguards of the appeals procedure, a diagnosis under the auspices of the Board's Advisory Committee on Occupational Chest Diseases (ACOCD).

In our view, the finding of an examining physician that an individual "may have or has" an asbestos-related condition fully warrants the attention of the ACOCD. We say this not only because such a finding signals that a diagnosis of asbestosis is possible but because of the existence, thanks to the Board's initiative, of the AFDE guideline. We fully recognize that this guideline is the product of a plant-specific programme and appreciate its several limitations as presented to us in testimony. The guideline is nonetheless a contribution to the earlier detection of lung fibrosis. Degrees of lung fibrosis are part of a continuum. As diagnostic procedures improve, it may be possible to detect fibrosis sooner, and the time at which a diagnosis of asbestosis is made may change. It is, therefore, highly appropriate that the ACOCD should have the opportunity to assess dust effect cases reported to the Board by examining physicians. In this connection, the Board should take steps to ensure that all examining physicians under the Regulation Respecting Asbestos are familiar with the terms of the AFDE guideline, the manner in which it was developed, and its limitations. This will make its own contribution to the common education of examining physicians who survey the health of asbestos workers.

We have outlined what we consider to be fair and orderly procedures for determining what individuals, among continuing workers who have encountered historical levels of asbestos dust exposure, might have asbestosis or show signs of dust effects. The question of whether such workers should be offered removal and rehabilitation remains. We continue to be mindful that removal will be from levels of exposure that are very low. Nonetheless, we are not inclined to cancel what the Regulation offers, not least because we are sensitive to the anxiety that disease labelling can produce in individuals. Ample though the medical uncertainty on the effects of removal may be, we believe that this option should be available on a case-by-case basis, subject to a strict right of worker refusal and to a right of appeal when removal is not made available as an option, to workers with asbestosis or signs of dust effects.

In exercising their right to accept or refuse an offer of removal, workers are entitled to know *to what level* of exposure they will be removed. We have pointed out that the silence of the Regulation on this matter invites repetition of one of the most controversial and damaging facets of the SRAP experience. In our view, the key to avoiding such repetition lies in the low exposure levels mandated by the Regulation. These are sufficiently low that they make the matter of removal to relatively lower as distinct from no occupational exposure an irrelevant question. What is relevant, in our judgement, is that section 7(1) of the Regulation specifies that an asbestos control programme shall be in effect wherever an assessment discloses ". . . that a worker is likely to inhale or ingest asbestos and that the health of the worker may be affected thereby. . . ." It follows that the removal of a worker should be to a position where, either with the same or another employer, an asbestos control programme does not apply. The Regulation should so stipulate.

With respect to the rehabilitation of removed workers, the SRAP experience gives us reason to contain our expectations, given especially the relatively older age of the workers likely to be involved. The SRAP turned out to be more a programme to compensate the socio-economic disability of medically impaired or potentially impaired workers than a successful rehabilitation initiative. As such, however, the SRAP offers a telling reminder of the desirability of compensating for socio-economic disability. In Chapter 13 we pointed out that the many ramifications of a dual system that seeks to compensate separately medical impairment and socio-economic disability are too vast, given our terms of reference and the limited submissions we have received on the subject, to permit us to formulate a judgement on the desirability of such a system. We can say, however, that the SRAP experience is testimony to some attractive features of a dual approach to compensation. Especially among older workers, needs arising from socio-economic disability can often be greater than the pensions that a mixed impairment/disability scheme of compensation permits.

We deliberate in a context in which it is by no means clear whether Ontario might adopt a dual compensation system and, if so, under what rules. In such a context, rehabilitation programmes have a place if only to continue to offer to certain asbestos-related workers a measure of income maintenance geared to their socio-economic circumstances. Once, with the passage of time, the number of asbestos workers whose employment dates back to the 1960s and 1970s has dwindled, the Workers' Compensation Board should review the matter of rehabilitation with the assistance of the Advisory Council on Industrial Disease Policy, whose creation we recommend in Chapter 12. We suggest that the time to launch such a review should be not less than ten years from now.

In light of the above considerations, we recommend that:

14.1 The sections of the Regulation Respecting Asbestos and the portions of its Code for Medical Surveillance which deal with the removal and rehabilitation of asbestos workers should be thoroughly revised so as to:

- ***(i) remove from examining physicians any role in determining whether asbestos workers are fit, fit with limitations, or unfit to work in an asbestos exposure;***
- ***(ii) direct examining physicians to inform the worker and the Workers' Compensation Board of any finding that a worker may have or has a condition resulting from exposure to asbestos;***
- ***(iii) vest an unequivocal right to refuse removal in the individual worker; and***
- ***(iv) specify that removal entails moving the worker to a position where an asbestos control programme mandated by the Regulation does not apply.***

We further recommend that:

14.2 The Workers' Compensation Board should:

- ***(i) take steps to ensure that all examining physicians under the Regulation Respecting Asbestos are familiar with the terms of the Asbestos Fibre Dust Effect guideline, the manner in which it was developed, and its limitations;***
- ***(ii) automatically set up a claim for any worker who, according to the finding of an examining physician, may have or has a condition resulting from exposure to asbestos;***

(iii) refer every such claim to the Advisory Committee on Occupational Chest Diseases (ACOCD);

(iv) offer the option of removal and rehabilitation to workers who are confirmed, either by the ACOCD or upon appeal, as exhibiting asbestosis or dust effects; and

(v) review, in not less than ten years, and with the assistance of the Advisory Council on Industrial Disease Policy, the continuing appropriateness of offering removal and rehabilitation to asbestos workers.

B. Outreach

The long latency of abestos-related conditions means that there will be a flow of compensable disease occasioned by past workplace exposures for at least the balance of this century. The size of this flow can be approximated, but under assumptions that are sufficiently tenuous to mean that any number amounts to no more than an educated guess. The actual outcome will only be revealed over time. We shall begin by addressing the matter of numbers. Whatever the flow of future disease from past exposure, it is incumbent in equity to ensure that as many cases as possible will be identified for compensation purposes. The Workers' Compensation Board, to its credit, has been conscious of the importance of identifying cases that are potentially compensable, and we shall review the Board's efforts in this regard. We shall then prescribe additional measures designed to strengthen the Board's efforts in the domain of outreach.

B.1 Future Disease from Past Workplace Exposure

There will be continuing disease resulting from past exposure of workers to asbestos during the 1940s, the 1950s, the 1960s, and the 1970s. This disease and death may not yet have reached its peak, and it may continue into the next century. Because asbestos fibre levels to which workers were exposed dropped substantially during the 1960s and especially the 1970s, we can expect that the incidence of asbestosis will decline first, followed by declines in the incidence of lung cancer and mesothelioma. It is difficult to predict how much disease will result in Ontario from past exposures. We are aware of three studies which have usefully attempted to forecast future disease resulting from past exposure to asbestos in the United States.[37]

Julian Peto, Henderson, and Pike have estimated that in the United States, exposure of workers to asbestos before 1965 will ultimately cause about 37,500 mesothelioma deaths and 112,500 lung cancer deaths, most of which (perhaps three-quarters) have yet to occur.[38] For their part, Nicholson et al. have estimated that total cancer deaths from past occupational exposure to asbestos in the United States will range from 8,700 per year in 1982 to almost 10,000 per year by 1990, and thereafter about 9,000 per year to the turn of the century, declining thereafter.[39] Meantime, Doll and Richard Peto have estimated that in the United States in 1978, perhaps 5% of lung cancer was attributable to occupational asbestos exposure, and that this exposure may account for between 1 and 2% of total cancer deaths, that is, for a range of 4,000 to 8,000 deaths per year.[40]

With the warning that the exercise should not provoke delusions of precision, it is a simple matter to apply the Doll and Peto percentages to Ontario. In this province, in 1980, there were 3,280 deaths from lung cancer and 14,451 deaths from all cancers. Accordingly, 5% of 3,280, or 164 lung cancer deaths could be attributed to occupational asbestos exposure; alternatively, 1 to 2% of 14,451, or a range of 145 to 290 Ontario deaths per year from all cancers might be attributed to this exposure. Such an attribution involves the assumption that in Ontario a similar proportion of the population experienced an occupational exposure to asbestos similar to that in the United States. In fact, however, it appears that a substantially lower proportion of the Ontario population has experienced occupational asbestos exposure than has been the case in the United States. In shipbuilding and manufacturing, this proportion is less than half that of the United States.[41] Therefore, it is likely an overestimate to attribute a range of 145 to 290 cancer deaths per year in Ontario to asbestos.

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[37] A fourth study of which we are aware is "Estimates of the Fraction of Cancer in the United States Related to Occupational Factors," prepared by the National Cancer Institute, the National Institute of Environmental Health Sciences, and the National Institute for Occupational Safety and Health, Washington, D.C., 15 September 1978. This widely publicized study, commonly referred to as the "Estimates Paper," has been thoroughly discredited by responsible critics and therefore requires no comment from us. This study estimated that 23 to 38% of future cancer in the U.S. would be attributable to occupational exposure to just six known carcinogens, asbestos being one. It has been particularly well analyzed by Richard Doll and Richard Peto in "The Causes of Cancer: Quantitative Estimates of Avoidable Risk of Cancer in the United States Today," *Journal of the National Cancer Institute (JNCI)* 66:6 (June 1981): 1240–1241, 1304–1308. Doll and Peto rejected the "Estimates Paper" as having overestimated the fraction of occupationally induced cancer by perhaps a factor of 10 and indicated that the paper has found no support in the research community.

[38] Julian Peto, Brian E. Henderson, and Malcolm C. Pike, "Trends in Mesothelioma Incidence in the United States and the Forecast Epidemic Due to Asbestos Exposure During World War II," in *Banbury Report 9: Quantification of Occupational Cancer*, eds. Richard Peto and Marvin Schneiderman ([Cold Spring Harbor, New York]: Cold Spring Harbor Laboratory, 1981), pp. 51–72.

[39] William J. Nicholson et al., "Cancer from Occupational Asbestos Exposure: Projections 1980–2000," in *Banbury Report 9: Quantification of Occupational Cancer*, pp. 87–111.

[40] Doll and Peto, "The Causes of Cancer: Quantitative Estimates of Avoidable Risk of Cancer in the United States Today," pp. 1242–1245.

But let us suppose that an estimated range that is half of this level is more realistic: 75 to 145 deaths per year from all cancers could be attributable to occupational exposure to asbestos in Ontario. The Ontario Workers' Compensation Board has reported that it allowed 20 asbestos-related cancer claims (including mesotheliomas) in 1980. Even the low end of the range of 75 to 145 is almost 4 times higher than the 20 claims compensated by the Board in 1980. Attempting to estimate present or future deaths from past occupational exposure to asbestos may amount to no more than educated guessing, but it strongly suggests that deaths, which by law are compensable, are going uncompensated.[42] Outreach measures are therefore necessary and important.

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[41] The study by Nicholson et al. (see note 39, *supra*) reported in its Table 3 that there were 4.5 million new shipyard workers in the U.S. during the 1940s, most of them employed during the war years. Assuming a 100% annual turnover rate as was observed in the U.S., the comparable number of entrants into Ontario shipbuilding would have been 40,000 workers. (See Chapter 6, Table 6.13 of this Report.) This is 0.9% of the U.S. figure. Given an Ontario labour force that is 4% as large as the U.S. labour force, the proportion of Ontario workers potentially exposed to asbestos in shipbuilding was less than one-quarter of the U.S. proportion for the 1940s. The U.S. experienced a much larger wartime shipbuilding boom than Ontario. In the post-war decades of the 1950s and 1960s, Ontario employment in shipbuilding ranged between 1 and 2% of the U.S. figure, so that the proportion of Ontario workers in shipbuilding during these decades was one-quarter to one-half that in the U.S.

In manufacturing of asbestos products, Statistics Canada data show that the number of Ontario workers during the 1950s and 1960s averaged around 2,000, which is less than 2% of the number of U.S. asbestos manufacturing workers reported by Nicholson et al. in their Table 4. The proportion of Ontario workers in asbestos manufacturing thus appears to be less than half that in the U.S.

[42] We are aware of an article, four of whose authors are Board physicians, which has argued that the number of claims compensated by the Board might actually reflect the true incidence of occupationally induced cancer in Ontario. This is not an epidemiological study but an argument that runs as follows: A compensation agency might, despite an outreach programme, have missed some occupationally induced cancers. However, that same compensation agency's policy of benefit of doubt, coupled with its practice of ignoring smoking habits, may mean that the number of claims actually compensated overestimates the true occupational incidence of the deaths involved. This overestimate thus cancels whatever shortfall arises from the claims missed by outreach measures. We choose not to comment on this sophistry, but observe that it is no excuse for failure to pursue outreach measures vigorously. The article to which we refer is Alan C. Chovil, William J. McCracken, Emerson C. Dowd, Charles Stewart, Dorothy F. Burton, and Douglas W. Dyer, "Occupational Cancer: Experience in Ontario," *Canadian Medical Association Journal* 125 (1 December 1981): 1237–1241.

B.2 The Board's Outreach Measures

In the matter of the Board's outreach role, Professor Barth testified that ". . . many [compensation] agencies either feel they don't have the resources or it's beyond their responsibility. That was not the case here in Ontario, and I think the WCB is to be praised for that."[43] Specifically in the realm of asbestos, the Corporate Board established an outreach programme on October 12, 1976.[44] The occasion was the Corporate Board's approval of the guideline covering gastrointestinal cancer in relation to asbestos exposure, but the outreach programme was intended to seek out victims of any asbestos-related disease with the exception of asbestosis. This exception, it was explained to us, was made on the assumption that the Chest Surveillance Programme of the Ministry of Labour would have identified the victims of this disease.[45]

As an initial step in the outreach programme, the Executive Director of the Board's Medical Services (then Rehabilitation Services) Division, Dr. William J. McCracken, addressed a letter to all the practising physicians in Ontario. This letter mentioned the latency of asbestos-related diseases, referred to the Board's guidelines, asked physicians ". . . to build a suspicion index into [their] history taking. . ." by inquiring into their patients' past occupations, and invited them to notify the Board of any cases whose nature suggested that a claim might be initiated.[46] One can wonder about how many busy practitioners actively read an evidently duplicated letter beginning with the salutation "Dear Doctor," but the attempt to alert the medical community in general was nonetheless genuine.

At a more robust and time-consuming level, the outreach programme generated a team consisting of the Board's Chest Disease Specialist, Dr. Douglas W. Dyer, and a representative of the Industrial Diseases and Dependants Section of the Claims Adjudication Branch. The team visited 32 companies which were deemed particularly likely to have had significant asbestos operations. In Dr. Dyer's words:

> When we would visit a company, we would speak to one of the senior officials, plant manager, member of personnel, and have a general discussion about the problems of asbestos-related disease, with particular interest to the malignancies. We would present to them copies of the guidelines and ask if they would search their personnel files to determine whether or not any

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[43] RCA Transcript, Evidence of Professor Peter S. Barth, 24 August 1982, Volume no. 57, p. 91.

[44] RCA Transcript, Evidence of Dr. Douglas W. Dyer, 16 July 1982, Volume no. 51, p. 91.

[45] Ibid., p. 107.

[46] The Workmen's Compensation Board, Ontario, Written submission to the Royal Commission on Asbestos, #69, 15 June 1981, Appendix 8.

> members of the organization, or past members of the organization, had evidence of having had any of the three diseases, or died from any of the three diseases.[47]

The three diseases to which Dr. Dyer referred were mesothelioma, lung cancer, and gastrointestinal cancer. After the Board, part-way through the programme of company visits, approved its guideline for laryngeal cancer in 1978, this disease was added to the interviewing team's list of requests.[48] According to Dr. Dyer's testimony, initial opposition from certain companies, notably Raybestos-Manhattan, was overcome, and most provided information.[49] Organized labour was made aware of the company visits, first through a Board letter to the Ontario Federation of Labour and subsequently through contacts with any individual unions identified in the course of the company interviews.[50]

Information conveyed to the Board concerning persons possibly afflicted with the diseases of interest was handled by the Claims Adjudication Branch. A claims adjudicator would try to establish a confirmation of the cause of death through the Registrar General. The Board would then try to contact the next of kin, asking if they wished to have a claim initiated. Although the number of contacts that were made is unknown, the Board through this procedure had set up, by July of 1980, 22 claims.[51] None of these involved mesothelioma.[52] It was while the outreach programme was underway, but as a distinct initiative, that the Board carried out the detective work which led to the compensation of the mesotheliomas which had afflicted the war-time gas mask workers in Ottawa.[53]

The gas mask episode sensitized the Board to the possibility of asbestos-related disease in other war-time industries. Accordingly, the outreach programme in 1979 and 1980 acquired a specific focus on the shipbuilding activity of the war years.[54] During the war, numerous corvettes and mine sweepers were constructed in Ontario, asbestos being applied to these vessels as a fire retardant and insulator. Some 400 mine sweepers were built in Toronto at the foot of Spadina Avenue, but here the outreach programme was frustrated by the fact that none of the companies involved remained in business. In the result, the programme was limited to team interviews with Collingwood Shipyards, which built warships in Collingwood and Thunder Bay, and with Port Weller Shipyards.[55] The 22 claims set up by the Board as a result of the outreach programme by July 1980 included the outcome of its venture into war-time shipbuilding. Of these 22 claims, 2 were ultimately granted by the Board, one was withdrawn, and 19 were denied.[56] We pursued the relatively large number of denials at our hearings. It is apparent that the Board had sought, and received, information on any workers who had contracted the diseases of interest, however briefly they might have been employed. Accordingly, the 19 denied claimants included some very short-term employees, the duration of whose exposure to asbestos would have fallen well short of those specified by the Board's guidelines. This affords at least one explanation for the volume of denials.[57]

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[47] RCA Transcript, Evidence of Dr. Douglas W. Dyer, 16 July 1982, Volume no. 51, p. 92.

[48] Ibid., p. 101.

[49] Ibid., pp. 95–96.

[50] Ibid., pp. 93–94.

[51] Ibid., p. 96.

[52] Ibid., p. 101.

[53] The Ottawa gas mask workers were the subject of an epidemiological study by Dr. Alison D. McDonald, whose findings are reviewed by us in Chapter 5. The Board's attempts to identify individual gas mask workers for compensation purposes succeeded in the allowance of three mesothelioma claims. See RCA Transcript, Evidence of Dr. Douglas W. Dyer, 16 July 1982, Volume no. 51, pp. 108–119.

[54] Ibid., p. 102. See also, Barth, *Workers' Compensation and Asbestos in Ontario*, p. 7.4.

B.3 Improving Outreach Efforts

What is the index of success for an outreach programme? An obvious — and ghoulish — index is the toll of industrial disease and death uncovered. According to this index, a large "body count" permits an outreach programme to be deemed a "success"; a low count spells the verdict "failure." We instinctively recoil from using this index. Ideally, the most successful outreach programme would be one that simultaneously gave cause to be satisfied that occupationally induced disease and mortality were not going uncompensated and that unearthed what in fact was a minimum toll of suffering and sorrow.

The outreach efforts exerted by the Board between October 1976 and July 1980 yielded, as we have seen, 2 compensated claims for asbestos-induced disease. In our view, this statistic permits neither a judgement of success nor one of failure. We appreciate what the Board's outreach programme has been trying to achieve and note with approval that the Board does not consider that its efforts have reached an end. According to Dr. Dyer's testimony, the Board, in the summer of 1982, was updating its list of asbestos-using manufacturing companies with a view to arranging further team interviews.[58] Outside the domain of manufacturing, we note with Professor Barth that the Board's team interviews have not sought out employers of workers who are users of asbestos-containing manufactured products (e.g., auto mechanics) or of workers involved in building maintenance or demolition. We also share Professor Barth's caution that such workers are ". . . likely to be employed in many small, decentralized establishments rendering any outreach programme costly and slow."[59] We direct attention, however, to the fact that significant concentrations of brake mechanics, as indicated in Chapter 6 of this Report, have been employed in large automotive shops in operations that involve a substantial volume of bevelling and grinding. Such large shops can be a readily feasible subject for outreach measures.

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[55] RCA Transcript, Evidence of Dr. Douglas W. Dyer, 16 July 1982, Volume no. 51, pp. 102–103.

[56] Ibid., p. 96.

[57] Ibid., pp. 99–100.

[58] Ibid., p. 104.

[59] Barth, *Workers' Compensation and Asbestos in Ontario*, p. 7.7.

What is to be done? We are not aware of outreach measures taken elsewhere that compare favourably with the Board's own programme. Professor Barth observed that ". . . a very major publicity effort in the latter part of the 1970s by the U.S. government to identify asbestos-injured workers in federally operated shipyards is acknowledged to have met with failure or at least very little response."[60] If we are convinced that more vigorous outreach measures are necessary, this is because we cannot pronounce ourselves satisfied that no or even little asbestos-related disease is going uncompensated. The lowest end of the range of educated guesses on the numerical size of asbestos-induced occupational disease is too many times the number of compensated Board cases to be anything but disturbing. It is likely true that no occupational carcinogen has been more widely publicized than asbestos; asbestos should therefore be a relatively "easy" subject for outreach measures.

The initial consideration we wish to address stems from the added publicity which the existence of this Commission gave to asbestos; it points to the need for outreach measures which are not left solely to the Workers' Compensation Board but involve the co-ordinated joint efforts of the Board and the Ministry of Labour. On the occasion of one of our informal hearings, open to any person giving notice of a desire to speak, we received Mr. Floyd Lefebvre of Cornwall, Ontario, a former employee of Domtar Construction Materials Limited in that city.[61] Mr. Lefebvre spoke to us about his own respiratory problems and of his status as an unsuccessful Board claimant who, although granted an appeal hearing, decided not to pursue the matter because of his health. More generally, Mr. Lefebvre spoke of the use of asbestos in the Domtar plant, where he had begun employment in 1951, and of the fact that he and his fellow workers were periodically examined by the mobile x-ray unit of the Chest Surveillance Programme in the 1950s and 1960s. He had compiled a list of names of fellow employees, including some who had died prior to retirement age, all of whom he believed would appear on mobile x-ray unit reports. Mr. Lefebvre submitted all these names to us.

We followed up Mr. Lefebvre's submission with staff enquiries to the Workers' Compensation Board and the Ministry of Labour. The Board confirmed that Domtar Construction Materials had been visited by an outreach team in July 1978. It could identify only 2 asbestos-related claims from employees of Domtar, one of them Mr. Lefebvre's and neither of them successful.[62] By date of March 18, 1983, we received from the Ministry of Labour a letter which reported its own pursuit of our enquiry in the following words:

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[60] Ibid., p. 7.8.

[61] RCA Transcript, Submission by Mr. Floyd Lefebvre, 5 July 1982, Volume no. 47(B), pp. 142–153.

> An official of the Occupational Health Branch met with the Manager of Occupational Health and Hygiene of Domtar to review the company personnel records for the persons listed by Mr. Lefebvre in his testimony before the Commission. The Ministry has found that several workers on the list have died and some of the deaths were attributed to various cancers. There were three cancers of the lung, one case of mesothelioma and one case of cancer of the bowels.
>
> Since this discovery, I understand that the employer has decided to submit workers' compensation claims for those workers with causes of death which could be attributable to exposure from asbestos. In addition, the Ministry itself will conduct a study of the mortality experience of the exposed population at Domtar. As a result, there may be other cases which could lead to compensation claims being filed with the Workers' Compensation Board at some future date.[63]

These words speak for themselves. The publicized existence of this Commission led a former asbestos worker to make an appearance which triggered a meaningful if limited outreach effort. What we take from this episode to be particularly telling is the pointed demonstration of the capability of the Ministry of Labour to contribute to outreach measures. The Ministry's files offer a rich source of information, covering several decades, on asbestos-using processes in many Ontario establishments. The mobile x-ray unit reports of the Ministry's Chest Surveillance Programme contain thousands of employee names. Clearly, the Ministry of Labour is equipped to play the role of a full participant in outreach efforts. It in no way belittles the officials who were involved to say that employer interviewing teams composed of a Board physician and a representative of the Board's Claims Adjudication Branch are painfully shorthanded. Ministry of Labour officials with Ministry information at their fingertips are necessary members of such teams. Interviews aside, we consider the Ministry essential to the success of new outreach initiatives, which we shall address below.

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[62] Letter from Mr. Andy Emmink, Assistant Secretary, Workers' Compensation Board to the Royal Commission on Asbestos, 7 January 1983.

[63] Letter from Mr. Arthur L. Gladstone, Senior Policy Advisor, Occupational Health and Safety Division, Ontario Ministry of Labour to the Royal Commission on Asbestos, 18 March 1983.

In our view, the logical starting point is to stipulate a new and joint Ministry-Board responsibility for outreach measures in formal terms. We have been informed by the Ministry of Labour of a Memorandum of Understanding, dated May 14, 1982, between the Minister of Labour and the Chairman of the Workers' Compensation Board on Ministry-Board relations.[64] This document states that the Memorandum, ". . . as amended from time to time, is the formal description of their relationship."[65] We consider it most advisable that the Memorandum of Understanding should be amended to give effect to a joint Ministry-Board responsibility for the development and implementation of outreach measures designed to promote the identification of potential claims for industrial disease compensation. We therefore recommend that:

14.3 The Memorandum of Understanding which constitutes the formal description of the operating relationship between the Ministry of Labour and the Workers' Compensation Board should be amended to recognize a joint Ministry-Board responsibility for the development and implementation of outreach measures designed to promote the identification of potential claims for industrial disease compensation.

We anticipate that pursuant to this undertaking, an invigorated set of employer interviews would be pursued and contact with unions expanded. In the exercise of the Ministry-Board responsibility for outreach, discrete efforts might be made to ask the members of management and labour to be alert to causes of disease and death among past employees who may be known to them. Beyond this, let us address some new outreach measures which look to future rather than past disease and death. Here, an important focus should be to infuse the individual patient-physician relationship with information.

Let us consider the thousands of asbestos-exposed workers who have been examined by the Chest Surveillance Programme. Dr. Peter L. Pelmear, Director of the Occupational Health Branch of the Ministry of Labour, testified that the number of asbestos workers examined dropped precipitously between 1980 and 1982, from approximately 14,000 to 8,000.[66] This is consistent with the secular and cyclical trends currently affecting asbestos-using industry. From the standpoint of outreach, however, it underlines the growing magnitude of a long-standing problem: the number of ex-employees who, once they leave asbestos-using plants, are no longer covered by the Chest Surveillance Programme and, as of 1982, by the medical surveillance measures prescribed in the Regulation Respecting Asbestos. Among asbestos employers, Dr. Pelmear testified, only Johns-Manville has encouraged ex-workers to attend the Ministry's mobile x-ray units when they are in the plant.[67] This is not to say that ex-workers of other plants would not be examined if they appeared at their old plant when the mobile x-ray units were present; it means, however, that no effort is made to notify former employees of a mobile unit visit. There is an exception outside the domain of fixed place industry: since the 1960s, the mobile x-ray units have regularly visited the union halls of Locals 58 and 95 of the International Association of Heat and Frost Insulators and Asbestos Workers, with a response rate estimated by Dr. Pelmear to be between 40 and 60%.[68] A possibility which is open to any ex-asbestos worker is simply to visit the Ministry's central Medical Service Chest Clinic facilities at 880 Bay Street in Toronto, where an examination would be given upon request. (This Clinic was formerly called the Chest Diseases Clinic and was located at 50 Grosvenor Street in Toronto.) Self-evidently, the remoteness of this possibility grows with the distance of an ex-employee's residence from Toronto.

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[64] "Memorandum of Understanding Between the Minister of Labour and the Chairman of the Workmen's Compensation Board," 14 May 1982, appended to letter from Mr. Arthur L. Gladstone, Senior Policy Advisor, Occupational Health and Safety Division, Ontario Ministry of Labour to the Royal Commission on Asbestos, 7 January 1983.

[65] Ibid., p. 1.

[66] RCA Transcript, Evidence of Dr. Peter L. Pelmear, 21 June 1982, Volume no. 42(B), p. 7.

Both because the historical locations of asbestos-related firms span the province from east to west and north to south, and because the mobility of individuals is unlimited, workers who acquired occupational exposure to asbestos in Ontario may be anywhere. At our hearings, we probed the possibility that ex-workers might be subject to misdiagnosis. With respect to mesothelioma, Dr. Alexander C. Ritchie, Professor of Pathology at the University of Toronto, testified that physicians training at the Toronto General Hospital would see mesotheliomas because these cases are referred to him by the Board. If those were subtracted, he doubted that even the Toronto General would get more than one mesothelioma in four years.[69] It follows that even physicians who have received specialty training at a major centre are unlikely ever to have encountered this industrial disease; its identification therefore rests on a combination of well-developed general diagnostic skills and a physician's sensitivity to occupation as a source of disease. As for asbestosis, Dr. Ritchie considered that a diagnosis of nondescript chest fibrosis would be likely in many communities, especially if the patient and physician had not explicitly pursued the possibility that the patient might have been exposed to asbestos in his occupational history.[70]

In our view, the records of the Chest Surveillance Programme offer a logical starting point from which to build a more informed patient-physician relationship for ex-asbestos workers. We appreciate the fact that the Ministry of Labour is currently engaged in a systematic attempt to computerize these records and correlate them with its separate records of dust exposure in the plants in which individuals worked.[71] The names of the individuals involved, together with the specific plants in which they were employed, permit the launching of an intensive search through employers and unions for residential addresses. As addresses become known, a promising outreach effort can be undertaken by mailing to each individual a plastic card with an accompanying letter. The letter should invite the individual to present his card whenever he visits a physician. The card would inform the physician that the patient was once under surveillance as an asbestos worker, alert the physician to be sensitive to any symptoms that might be indicative of asbestos-related disease, and invite the physician to contact the Ministry or the Board (which of these two agencies might be contacted should have been jointly determined by the Ministry and the Board) for records of his patient or other pertinent assistance he might wish to have. Accordingly, we recommend that:

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[67] Ibid., p. 21.

[68] Ibid., p. 22.

[69] RCA Transcript, Evidence of Dr. Alexander C. Ritchie, 14 July 1982, Volume no. 49, pp. 85–86.

[70] Ibid., pp. 58–59.

14.4 The Ministry of Labour and the Workers' Compensation Board should attempt systematically to locate the residential addresses of all individuals who, because of previous asbestos exposure, have been examined by the mobile x-ray units of the Chest Surveillance Programme so that these individuals may be sent a card which they would be invited to present to their physicians whenever they wished. The card would inform the physician that its bearer was once under surveillance as an asbestos worker, alert the physician to be sensitive to any symptoms that might be indicative of asbestos-related disease, and invite the physician to contact the Ministry or the Board for records of his patient or other pertinent assistance.

We do not expect that the outreach measure just prescribed will be more than partially effective. Bearing in mind that many individuals will never be located and that others, especially construction workers, were never under surveillance, there is a need to address Ontario physicians generally. This, as we have noted, the Board did by sending a letter to every practising physician in November of 1976. A more systematic attempt to alert medical practitioners by a variety of means should be developed by the Ministry and the Board in consultation with organizations like the Ontario Medical Association and the Ontario Chapter of the College of Family Physicians of Canada. The importance of taking occupational histories and of being sensitive to possibly compensable disease is such that, personal correspondence aside, these matters are eminently fit subjects for local district council meetings of practising physicians and for recurring notes and articles in professional publications. We therefore recommend that:

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[71] RCA Transcript, Evidence of Dr. Peter L. Pelmear, 21 June 1982, Volume no. 42(B), pp. 8–9.

14.5 The Ministry of Labour and the Workers' Compensation Board should systematically seek the co-operation of organizations such as the Ontario Medical Association and the Ontario Chapter of the College of Family Physicians of Canada to encourage all practising physicians to take work histories of patients whenever possible symptoms of asbestos-related disease are suspected and to inform such patients on how to initiate compensation claims. The Ministry and the Board should encourage these organizations to spread an understanding of the state of occupational medicine among physicians by all appropriate means, including local district council meetings and professional publications.

Finally, there is the institutional role of hospitals in outreach measures. This role has yet to be exploited. It could be made a matter of hospital policy that occupational histories will be solicited when individual patients are admitted to hospital with symptoms of diseases that might be asbestos-related. The individuals most likely to be helpful in this regard are the chief executive officers of Ontario hospitals. We suggest that the Ministry and the Board should approach these officials with the co-operation of the Ontario Hospital Association. Accordingly, we recommend that:

14.6 The Ministry of Labour and the Workers' Compensation Board, with the co-operation of the Ontario Hospital Association, should communicate to the chief executive officers of Ontario hospitals the importance of taking steps that will promote, as a matter of hospital policy, the soliciting of occupational histories from patients who are admitted to hospital with symptoms of disease that may be asbestos-related.

We consider that the above prescriptions provide means which should enhance the effectiveness of outreach measures initiated by the Board in 1976. Whatever the means, the question of what constitutes a successful outreach programme remains. As already stated, we do not deem the numerical toll of disease and death uncovered to be an appropriate index of success or failure. Rather, the overriding criterion is the judgement that occupationally induced disease and mortality are not going uncompensated. This judgement is not easily made because it must take many factors into account — from ongoing epidemiological findings to industrial structure. And it is not a once-and-for-all judgement because it rests on learning from ongoing outreach experiences and must be open to suggestion. These considerations lead us to the view that the Advisory Council on Industrial Disease Policy, whose creation we have prescribed, is an eminently appropriate vehicle to assess periodically the effectiveness of the outreach efforts undertaken by the Ministry and the Board. This Council, with its general mandate to advise on industrial disease policy, can bring collective judgement to bear openly on the relative success of outreach measures. We therefore recommend that:

14.7 The mandate of the Advisory Council on Industrial Disease Policy should include the role of monitoring the outreach efforts of the Ministry of Labour and the Workers' Compensation Board in an ongoing manner, assessing the relative success of these efforts, and advising on appropriate means of enhancing outreach.

C. Prevention

C.1 Introduction

The primary purpose of any workers' compensation system is, of course, to compensate workers who have suffered occupational illness or injury. But it has long been recognized that such a system can also play a role in prevention. Thus, for example, the Ontario Workers' Compensation Board has long had a statutory role in promoting, approving, and funding accident prevention associations. (See Chapter 8.) Given the *Occupational Health and Safety Act,* primacy in accident and disease prevention is clearly accorded to the Ministry of Labour in concert with the Internal Responsibility System, but it is equally clear that the Board is expected to play a supportive role.

How can the financing of workers' compensation support the objective of accident and disease prevention? To ponder this question is to reflect not only upon prevention but upon other objectives as well, notably equity and justice. In the financing of workers' compensation, equity will require, as a general proposition, that similarly situated employers will be treated similarly. Just as the objective of prevention indicates that the financing of workers' compensation might include incentives to reduce injury and disease, the objective of equity indicates that an employer whose working conditions lead to disproportionately high claims should finance some or all of the extra compensation burden that his activities impose. Finally, justice, in the sense of retribution, is an objective that might be accommodated within a compensation system. While the appropriateness of revenge can be questioned as a social goal, the example that is set for others by retributive justice can make its own contribution to prevention.

Recent newspaper accounts of workplace tragedies have included statements by injured workers or their next of kin demanding that a penalty be imposed upon the employer, not because this would enrich the injured worker or his survivors, nor spare the other contributors to the system, but rather because the employer deserves to be punished. Testimony before this Commission has included impassioned pleas that certain employers whose places of work are the origin of considerable illness and death be made to account for the suffering that they have caused.[72]

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[72] See, for example, RCA Transcript, Submissions by the Asbestos Victims of Ontario, 16 February 1981, Volume no. 1, pp. 142-190.

These objectives may be pursued within the framework of a workers' compensation system, or outside that framework. In fact, workers' compensation itself replaced a system of tort liability which served, to some extent, purposes of compensation, prevention, equity, and retribution. Workers' compensation provided workers a no-fault guaranteed degree of protection from losses due to industrial injury and disease in lieu of the common law right of these workers to sue their employers in court. The extent to which the Ontario compensation system effectively bars the exercise of common law rights is substantial and clearly greater than in the United States. In most American jurisdictions, the worker, while barred from suing his employer, may bring suit against third parties, for example, suppliers of asbestos used in the workplace. In Ontario, such third-party suits are in effect precluded because no scheduled firm, under the terms of the *Workers' Compensation Act*, may be sued, whether as the employer or as a supplier.[73] It is also possible in some American states, but not in Ontario, for an employee to bring suit if the conduct of the employer in exposing the employee to risks was wanton or willful. More generally, the tort system in the United States applies a standard of strict liability; in Canada the plaintiff must prove that the defendant was negligent, an added burden on the plaintiff. These differences account for the enormous volume of asbestos-related litigation in the United States and its virtual nonexistence in Ontario. Have we reached a time when the compensation system should be supplemented, at least in part, by tort liability for workplace-related diseases? Are asbestos-related diseases a special case which should involve special rights for private lawsuits? After due deliberation, we have concluded that we must answer both of these questions in the negative.

Tort liability is an unattractive addition to workers' compensation because, first, it is an inefficient means of compensating victims of industrial disease. We need only look to the experience in the United States with asbestos-related lawsuits over the last decade to see how inefficient the system actually is. It has been reported that before the Manville Corporation (successor to the Johns-Manville Corporation in the United States) filed for bankruptcy in August of 1982, defence attorneys were charging the company about $2 million per month, which amounted to more than half the $40,000 per case average cost of disposing of claims.[74] If each claim cost Manville $40,000, half of which was paid to defence lawyers, that left no more than $20,000 to be paid to the injured worker. Many U.S. claims are brought by lawyers who are paid a contingent fee of one-third or more of the recovery. If the plaintiff's lawyer took one-third of the $20,000, that would leave the injured worker with $13,300, or one-third of the amount expended by Manville on the average claim. This can be compared to the experience of the Workers' Compensation Board in Ontario where less than 9% of all revenue is paid out for administration, and there is virtually no litigation.[75] In fact, the contrasting costs of administering the asbestos litigation in the United States are even higher than our figures indicate, because in addition to the claims by workers against manufacturers such as Johns-Manville, there is tremendous litigation among the defendant manufacturers and their insurance companies to determine who will ultimately pay the damages that are awarded. Commentators have observed that the U.S. asbestos litigation benefits primarily the lawyers, and not the injured workers.[76] The huge number of asbestos-related cases now pending in the courts in the United States[77] have threatened to swamp the capacity of that legal system in some jurisdictions, greatly increasing delays for other lawsuits and of course consuming enormous resources in judicial time.

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[73] Where a third party is not a scheduled employer in Ontario, s. 8(1) of the *Workers' Compensation Act* (R.S.O. 1980, c. 539) permits an employee to elect to bring an action. In addition, a recent case held that an employee could sue an *executive officer* of the employer in certain circumstances. See *Berger v. Willowdale A.M.C. et al.* (1983), 41 O.R. (2d) 89 (Ont. C.A.).

[74] James S. Granelli, "The Future Claims Fight," *The National Law Journal* 5:30 (4 April 1983): 28.

Second, private lawsuits by workers for industrial disease are unattractive because, in general, they are difficult for the worker to win. The worker must first prove that the work exposure caused his illness. This requires demonstrating that the employee was exposed to the substance in question and perhaps showing the extent of exposure. Even with asbestos-related diseases such as mesothelioma and asbestosis, if the worker had been employed in several jobs involving asbestos exposure, he might have difficulty proving that any particular defendant had caused this particular disease. Far worse is the case of lung cancer, which may be a result of asbestos exposure, smoking, dietary or other causes. It may be extremely difficult to convince a court that a particular lung cancer case was caused by asbestos exposure. While numerous asbestos victims have succeeded in recovering in U.S. lawsuits, the success rate may decline as the more difficult cases reach trial.

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[75] Paul C. Weiler, *Reshaping Workers' Compensation for Ontario*, a report submitted to Robert G. Elgie, M.D., Minister of Labour ([Toronto: Ontario Ministry of Labour], November 1980), p. 20.

[76] See, for example, David Lauter, "Footing the Bill for Toxic Torts," *The National Law Journal* 5:21 (31 January 1983): 1, which suggested that only $400 million of the $2 billion that the Manville Corporation might pay in claims would reach asbestos victims, the rest going to lawyers, experts, and court costs. The litigation cost, said an insurance company officer, "is an embarrassment to us." Former Representative Millicent Fenwick (R-NJ) wrote a letter to *The New York Times* saying, "A lawsuit — is expensive and time-consuming. If an award is ever granted, it is seriously depleted by the fee charged by the worker's lawyer, and often the suit is never resolved within the victim's lifetime." *The New York Times*, 10 March 1983. *The Chicago Tribune* stated in editorial: "The chief beneficiaries are not the claimants, they are the trial lawyers." "The Asbestos Dilemma," *The Chicago Tribune*, 1 September 1982, Editorial. *The Washington Post* stated editorially: "The present system is serving no one but — who else? — the lawyers." "The Asbestos Mess," *The Washington Post*, 27 April 1982, Editorial.

[77] In August 1982, there were 16,500 claims pending against Johns-Manville alone, and 500 per month were being filed, according to a Manville advertisement in *The Wall Street Journal*, 27 August 1982.

A third problem is the time delays involved in civil litigation. Years may pass between the worker filing a claim and his receipt of an award. During this time, the worker may be under serious financial pressure and be tempted to settle his case immediately for a small fraction of what he might ultimately be able to win. These delays, combined with the great uncertainty about the final outcome of most cases, are a serious deterrent to workers exercising the right to sue.

Finally, there is the problem of collecting any damages that are awarded by the court. While this may not be a problem in typical injury cases, it is potentially a serious problem in the case of long-latency diseases, such as the asbestos-related diseases. If the disease is not manifest for 20 or more years after exposure, and a lawsuit is not completed for many years after that, there is always the possibility that the employer will be out of business, out of the jurisdiction, or bankrupt. If the employer has left the jurisdiction, it may be possible to enforce a judgement in foreign jurisdictions, but this may be costly and time-consuming. If the employer has gone out of business, then there is no one for the employee to sue. Of immediate relevance to asbestos litigation is the possibility that the employer might file a bankruptcy petition as the Manville Corporation has done in the United States. The legal effects of filing may not be resolved by the courts for years. Even if claims may continue to be brought against the assets of the reorganized company, the reorganization may limit the total amount payable to those claimants, as is the primary purpose of the Manville filing under Chapter 11 of the U.S. *Bankruptcy Act*. The receiver has a fixed pool of assets to distribute to all claimants, and as the total of asbestos-related lawsuits increases, the amount each individual can receive diminishes since they are drawing upon a fixed pool. In any event, the maneuver of filing for bankruptcy is further lengthening the already time-consuming process of effecting settlements.

In their study for this Commission, Professor Carolyn J. Tuohy and Professor Michael J. Trebilcock reviewed tort liability and found it seriously deficient for dealing with occupational disease.[78] We are impressed by the litany of problems that a civil liability regime would bring and by the uncertain benefits, given the availability of bankruptcy proceedings for a beleaguered defendant such as Johns-Manville. The uncertainty, the legal costs, time delays, and burden upon the court system all lead us to reject the creation of a right for injured workers to sue their employer for redress of those injuries.

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[78] Carolyn J. Tuohy and Michael J. Trebilcock, *Policy Options in the Regulation of Asbestos-Related Health Hazards*, Royal Commission on Asbestos Study Series, no. 3 (Toronto: Royal Commission on Asbestos, 1982), chaps. 7 and 10.

Is creation of a tort right of action for injured workers necessary to provide adequate compensation to victims of industrial disease? We think not. With respect to other jurisdictions where injured workers have a right to sue their employer, such as England, Professor Barth testified that the level of compensation benefits is lower than what it would otherwise be on the grounds that those most seriously injured have another source of compensation.[79] As for the general adequacy of current workers' compensation benefits in Ontario, this is currently under review in the wake of Professor Paul C. Weiler's inquiry, posing questions which lie well beyond our terms of reference but signalling possible avenues of improvement. In the present setting, we join Professor Weiler in the conclusion that reviving a tort right of action would be a "retrograde step."[80]

Having decided that the compensation objective is best met within the workers' compensation system, and having rejected the creation of the right to sue by injured workers, we must now explore the workers' compensation financing system in relation to the other objectives discussed above. How well does workers' compensation serve the objectives of prevention, equity, and retributive justice? Here we face a range of tradeoffs that have been made between the principle, inherent in workers' compensation, of collective insurance of employers and the incentive to provide safe and healthy workplaces. There is also a tradeoff between the objective of collective insurance and that of equity among employers bearing the burden of compensation claims. Pursuing the prevention and equity objectives is, as we shall see, especially difficult when we are dealing with diseases of long latency, which is precisely the type of disease that is associated with asbestos.

We shall now describe the current system of financing workers' compensation, examine statutory means of enhancing the Board's role in prevention in an equitable and just manner, and make certain observations which should receive attention in reforming the financing of workers' compensation.

C.2 The Current Financing of Workers' Compensation

The Ontario workers' compensation system collects revenue from Ontario employers by levying an assessment on the payroll of each employer. The 150,000 or more employers under Schedule 1 of the *Workers' Compensation Act* are classified into 108 industry rate groups. A rate group may have between 1 and 24,000 members. The assignment to rate groups is designed to place employers who produce similar products or provide similar services into the same rate group on the presumption that they have the same accident or disease risks. All employers in a given industry rate group pay the same assessment rate based on their assessable payroll. The average assessment is under 2% of payroll, but ranges from a low of 0.2% for the accounting rate group to over 16% for loading and unloading cars.[81] The rate for a given rate group is changed annually to reflect the percentage change in the claims experience of the group from one year to the next. A single firm with several operations may have its operations classified in different rate groups. Thus, at the beginning of 1980, Johns-Manville Canada appeared in five separate rate groups. The asbestos-cement pipe plant in Scarborough was in rate group 137: "Bricks, Blocks and Tile." The insulation plant, also in Scarborough, was in another rate group.

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[79] RCA Transcript, Evidence of Professor Peter S. Barth, 24 August 1982, Volume no. 57, p. 51.

[80] Weiler, *Reshaping Workers' Compensation for Ontario*, p. 14.

Within a rate group, firms may be "experience rated" if a majority of the members of the group vote in favour of experience rating. If a group is experience rated, then for each firm, its three-year average accident cost rate per $100 of payroll will be compared to that of the entire rate group. If the firm's cost rate is lower than that for the group, the firm may receive a credit of 25% or 50% of the difference between its cost rate and the group's cost rate, multiplied by the assessable payroll for the last year. If the firm's cost rate is greater than that of the rate group, a surcharge of 25% or 50% of the difference will be made. The rate group can choose whether it wishes to be 25% or 50% experience rated. There are other limits on the extent to which a firm's rating can be modified by the experience rating plan. Importantly, for our purposes, the experience rating credit or surcharge is always a fraction of the firm's assessable payroll.[82]

The financing system described above is based on multiplying the assessable payroll of a firm times some percentage. If the assessable payroll drops to zero, then the firm's contribution to the Board's accident fund also drops to zero. Since a firm may have its operations classified into more than one rate group, it is possible for a firm to continue to do business in Ontario, yet cease to contribute to the fund on behalf of one of its operations. For example, Johns-Manville Canada ceased operation at its pipe plant in Scarborough in 1980. The moment that the plant was closed and the payroll terminated, Johns-Manville's contribution to the accident fund on behalf of its employees of that plant also dropped to zero. In 1981, claims, including disease claims attributed to the Scarborough pipe plant, totalled $831,000. Receipts from Johns-Manville in respect of that pipe plant were zero, as were experience rating charges.[83]

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[81] Ibid., p. 81.

[82] The information in the preceding two paragraphs is drawn from letter and attachments from Mr. Alex Joma, Secretary, Workmen's Compensation Board to the Royal Commission on Asbestos, 22 June 1982.

Tragically, what we have categorized as the world-class occupational health disaster at this plant has yet to run its course. We can, therefore, expect a continuing volume of claims for asbestos-related disease due to exposure at this plant to be filed over the next decade or so. However, given the prevailing financial system, the Board will continue to record claim costs attributable to that plant and continue to record zero receipts unless the pipe plant is reopened and the payroll rises above zero. On the same property, Johns-Manville continues to do business in its insulation operations, and its other businesses in the province also continue. Similarly, when the Bendix brake operations in Windsor terminated, the relevant payroll dropped to zero as well. Asbestos-related claims filed after the termination will be recorded on the file of that plant at the Workers' Compensation Board, but there will be no contributions from Bendix to finance those claims.[84] Claims which are paid after an employer has left a rate group are borne by the remaining members of that rate group, unless the Board determines that this imposes an unreasonable burden on these employers, in which case they will be borne by the accident fund generally.[85]

The question of how well the prevailing rating system works for industrial accidents is beyond our terms of reference, but this system presents obvious deficiencies with respect to long-latency diseases. When there is a long time delay between the worker's exposure and the filing of a claim (and this delay is often 20 years or more with asbestos-related diseases), there is considerable opportunity for the employer to avoid paying for that disease when it finally appears. If an employer realizes that a large volume of claims are likely to be filed because of activities several decades previously, and if the employer accounts for a large proportion of the claims in his rate group, or the group is experience rated, there is an obvious incentive to avoid paying for the claims by reducing payroll in the activity that generated those claims and by not resuming employment activities. It is no small matter for a firm to decide to close a profitable plant. Still, one can imagine a situation where the expected workers' compensation assessments, including experience rating if it is applied, might loom sufficiently large to encourage an employer to close the plant permanently. This generates inequity among employers, because either the remaining members of the rate group, or all employers through the accident fund, must carry the expenses caused by the departed member. Equally serious, the objective of prevention is circumvented, since the employer can avoid even sharing these costs with other members of the group and also has nothing to fear from retribution. The contrast between the financial liability for industrial disease faced by Johns-Manville in Ontario and Johns-Manville in the United States is stunning. In addition to whatever workers' compensation payments Johns-Manville has made in the United States, it has faced lawsuits which it considered so crushing that it filed for reorganization and bankruptcy in 1982. In Ontario, between 1971 and 1981, the company paid the normal assessment for rate group 137 plus experience rating charges amounting to a total of $2.459 million. Total workers' compensation claims from the Scarborough Johns-Manville plant over that decade imposed costs of over $6.087 million. The Workers' Compensation Board calculated that if the company had been 100% experience rated, it would have paid $8.285 million to the Board. Thus, there is a deficit from this plant alone of $5.826 million,[86] which was to be borne by other members of rate group 137 until the Board, in October 1982, assigned the deficit to the accident fund where it is borne by all Ontario employers.[87] In short, Johns-Manville has borne virtually none of the costs of the disease it has occasioned in Ontario, while it was facing a staggering cost for its involvement in similar diseases in the United States.

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[83] RCA Exhibit IV-5, in RCA Transcript, Evidence of Mr. Alan G. MacDonald, 11 August 1982, Volume no. 55, p. 128: The Workmen's Compensation Board, Ontario, Inter-Divisional Communication from Mr. John C. Neal, Board Actuary to Mr. Alan G. MacDonald, Vice-Chairman of Administration and General Manager, 9 July 1982.

[84] RCA Transcript, Evidence of Mr. Alan G. MacDonald, 11 August 1982, Volume no. 55, pp. 119–120.

[85] *Workers' Compensation Act*, R.S.O. 1980, c. 539, s. 108(1).

C.3 Recovering from High-Claims Employers

The *Workers' Compensation Act* includes a provision allowing the Board to collect revenue from an employer with an unusually high claims rate. Section 91(7) of the Act provides as follows:

> Where the work injury frequency and the accident cost of the employer are consistently higher than that of the average in the industry in which he is engaged, the Board, as provided by the regulations, may increase the assessment for that employer by such a percentage thereof as the Board considers just, and may assess and levy the same upon the employer . . . .

Levies made under this section are referred to as "penalty assessments" and are governed by section 6 of Regulation 951 made under the Act, which provides as follows:

> 6. (1) The increase of assessment that the Board may levy under subsection 91(7) of the Act shall be levied where an employer within an individual rating classification,

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[86] RCA Exhibit IV-5, The Workmen's Compensation Board, Ontario, Inter-Divisional Communication from Mr. John C. Neal to Mr. Alan G. MacDonald.

[87] "WCB Fees Raised Over Asbestos Claims," *The Globe and Mail*, 18 October 1982.

> (a) has incurred in two of the last three complete years of operation a deficit accident cost experience, including his proper share of administration, safety and other expenses;
>
> (b) has incurred a lifetime deficit accident cost experience, including his proper share of administration, safety and other expenses; and
>
> (c) has incurred during two of the last three complete years of operation a frequency rate of compensable accidents at least 25 per cent higher than the average rate in the industry in which he is classified.
>
> (2) The actual payroll for the last complete year of operation under review shall be the basis for any additional assessment to be levied under subsection (1).
>
> (3) The first increase in assessment under subsection (1) shall be 100 per cent of the assessment based on the individual rating classification of the employer.
>
> (4) The amount of increase on any subsequent increase in assessment under subsection (1) shall be in the discretion of the Board.[88]

When the three conditions of section 6(1) in Regulation 951 have been met, then the Board may levy a penalty assessment which, in the first year, must be equal to the regular assessment for the last complete year, and in subsequent years can be any amount in the discretion of the Board. Despite the fact that the Scarborough Johns-Manville pipe plant led to claims at the Workers' Compensation Board during the decade 1971 through 1981 which generated a deficit for that plant of $5.826 million, no penalty assessment has ever been levied against Johns-Manville. The three conditions specified in section 6(1) of Regulation 951 were not met until the year 1980, because until 1978 Johns-Manville's accident frequency was less than the rate group average. But in 1980, the pipe plant in Scarborough was closed, and it is apparently the Board's policy not to levy a penalty assessment on a plant after it has closed.[89] If a penalty assessment had been made for the year

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[88] R.R.O. 1980, Reg. 951 under the *Workmen's Compensation Act*.

[89] RCA Exhibit IV-5, The Workmen's Compensation Board, Ontario, Inter-Divisional Communication from Mr. John C. Neal to Mr. Alan G. MacDonald. The following statement appears in this Exhibit under the heading Canadian Johns-Manville — 91(7) History: "This firm met the three basic conditions for a 91(7) for the three years 1978 to 1980, but it was not levied since Johns-Manville discontinued operations under firm and rate combination in May 1980."

1980, the amount would have been specified by section 6(3) of Regulation 951 as 100% of the assessment based on the individual rating classification of the employer. In 1980, the assessment of the pipe plant was $60,985, so the penalty assessment for that year would have been precisely this amount.[90]

Can the Workers' Compensation Board now levy a penalty assessment under section 91(7) of the Act for 1980, even though the pipe plant is closed? The Board apparently believes that this is either impossible or inappropriate, but we find nothing to sustain this view. Because the Johns-Manville plant did not close until May 1980, it still had a payroll during that year. In our view, failing to levy an assessment in the case of Johns-Manville sends a perverse message to other employers in the province that even a disastrous worker health experience need not lead to the exercise of section 91(7). Such a message destroys any incentive that might otherwise exist through the workers' compensation system to protect the health of workers. We see nothing in the language of section 91(7) of the Act or of section 6 of Regulation 951 which would preclude the application of a penalty assessment to Johns-Manville for the year 1980 even at this time. The employer, Johns-Manville Canada, is still doing business in the province and is available to pay a penalty assessment. We therefore recommend that:

14.8 The Workers' Compensation Board should levy against Johns-Manville Canada the maximum penalty assessment that can be made under section 91(7) of the Workers' Compensation Act in respect of the operation of the asbestos-cement pipe plant in Scarborough which, in 1980, was a member of rate group 137.

This penalty assessment, if applied for 1980, would collect approximately $60,000, which is only 1% of the $5.8 million deficit of revenues from the pipe plant subtracted from the revenues that would have been collected if the firm were 100% experience rated. Equity among employers, to say nothing of the prevention objective, will not be achieved by such minor penalties.

The subject of penalties is one which we believe can and should be addressed separately from the matter of experience rating. Obviously, the latter can serve the prevention and equity objectives, although the insurance function of workers' compensation is also an important one, not to be dismissed lightly. We shall address the appropriateness of applying experience rating to industrial disease later in this chapter. Special penalties are a different matter, because they serve retributive justice, and through this objective also serve the goals of prevention and equity. In the realm of industrial disease, there exists in our view a circumstance in which a substantial volume of disease claims cries out for justice. This circumstance exists where the adverse health claim experience is the result not of chance but of a firm having exposed its employees to a serious health risk without divulging to them what the firm knew about the nature and magnitude of that risk. We trust that this circumstance has been unusual in Ontario in the past and will be truly exceptional in the future. Because this Commission was not established to determine past wrongdoing, we have not investigated and do not pass judgement upon any knowledge that Johns-Manville Canada may or may not have possessed about the health risks that its workers faced in the 1950s and 1960s, although we observe that American courts have held that certain asbestos employers had knowledge at or before this time.[91] The point remains, as a general proposition, that the possibility of retributive justice is at its most desirable in exceptional circumstances.

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[90] Ibid., Appendix A, p. 4.

We consider that the *Workers' Compensation Act* should be amended to permit the Board to serve the interests of retributive justice in the circumstance we have outlined. This should involve not a penalty assessment by the Board, but instead, given the seriousness of withholding knowledge about health hazards from workers, a statutory right of the Board to take action against an employer whom it believes has withheld such knowledge from its employees. The seriousness of this matter is such that its determination is best left to the courts. The Board should be able to invoke this statutory right of action whenever an employer's disease record has become sufficiently serious to result in claims whose value exceed its contributions to the workers' compensation system, however experience rated. So as to focus the attention of the litigants and the courts solely upon the matter of employer knowledge, and thereby circumvent the time-consuming matter of assessing damages, we believe that the statute should fix the amount that could be recovered by the Board in a successful action. We suggest that this amount should be fixed at twice the amount paid out on behalf of the employer's disease claims, one-half to replenish the accident fund, the other half to be apportioned by the Board as an additional award to its claimants.

In line with the above considerations, we recommend that:

14.9 The Workers' Compensation Act should be amended to give the Workers' Compensation Board a statutory right of action against any employer the value of whose disease claims exceeds his contributions to the accident fund when the Board has reason to believe that the employer withheld from his employees knowledge which he possessed about the hazardous nature of a substance to which he exposed them. The amount of recovery by the Board should be statutorily fixed as

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[91] For example, *Borel v. Fibreboard Paper Products Corporation*, 493 F.2d 1076 (1973), cert. denied 419 U.S. 869 (1974); and *Karjala v. Johns-Manville*, 523 F.2d 155 (1975).

twice the value of the employer's disease claims occasioned by the substance in question, one-half to be paid into the accident fund, the other half to the claimants involved.

C.4 Prevention and the Financing of Workers' Compensation

The statutory right of action that we have prescribed for the Board in exceptional circumstances should exist independently of whatever general framework is in place for financing workers' compensation. As for this framework, Professor Weiler has addressed the outline of what it might contain. In his 1980 report, he recommended that more extensive use be made of experience rating and addressed the problem of ensuring that a firm discharges its obligations to the compensation fund if it leaves the industry or the province.[92] In his 1983 report, he again endorsed the concept of experience rating, but suggested that long-latency diseases may not appropriately be part of any experience rating programme.[93] The Board is currently considering a universal system of experience rating that might be more aggressive than the current system.[94]

We wish to address the appropriateness of experience rating with respect to long-latency diseases, of which all asbestos-related diseases are a classic case in point. We join Professor Weiler in his observation that experience rating for such diseases will likely not generate substantial incentives for prevention given the number of years, equivalent to a generation, that separate a firm at the time of the exposure from that same firm at the time disease occurs. We must point out, however, that whatever the limitations of experience rating with respect to prevention, such rating preserves horizontal equity among employers once long-latency disease materializes. As we have described it, the Johns-Manville experience as a member of rate group 137 seriously offends the principle of horizontal equity.

In time, the asbestos episode is one that will recede into history. The current occupational health regime, as embodied in the *Occupational Health and Safety Act*, requires the disclosure of hazardous substances, provides for their designation, and mandates their regulation. Such disease risk as may remain should be sufficiently low that experience rating might well be reduced, or even eliminated, in deference to the principle of collective employer insurance. We are content to leave this debate to others.

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[92] Weiler, *Reshaping Workers' Compensation for Ontario*, chap. 3.

[93] Paul C. Weiler, *Protecting the Worker from Disability: Challenges for the Eighties*, a report submitted to Russell H. Ramsay, Minister of Labour ([Toronto: Ontario Ministry of Labour] April 1983), pp. 129–130.

[94] Ibid., p. 120.

However, we feel bound to observe that the phenomenon of long-latency disease is precisely such that, for at least the next two decades, asbestos-related disease claims will be related to industrial exposures that antecede the current occupational health regime. In this setting, experience rating can of course do nothing to prevent past exposure but has major implications for equity among employers. This speaks for a situation in which, however the Board might contemplate revising the experience rating of industrial disease generally, it should consider the financing of asbestos-related claims, at least to the turn of the century, as a special case. The principle of horizontal equity speaks for greater rather than less experience rating than the minimal degree which applies at present. Accordingly, we recommend that:

14.10 In approaching the matter of any revisions in the experience rating of industrial disease, the Workers' Compensation Board should consider the financing of asbestos-related claims, until at least the turn of the century, as a special case. The Board should be sensitive to the proposition that the principle of horizontal equity speaks for greater rather than less experience rating among asbestos employers, and at a minimum does not countenance less experience rating than applies at present.

Professor Weiler has posed an alternative to experience rating for long-latency disease resulting from future exposure that may simultaneously serve the goals of prevention and equity. This alternative is the exposure charge, which is a payment made by an employer based upon the extent of employee exposure to hazardous substances and paid at the time of exposure, rather than at the time the disease develops.[95] The revenues from such a charge could be paid into the compensation fund, and thereby reduce the burden upon employers who do not expose their employees to hazardous substances, while increasing the financial burden on those who do. This serves the equity objective. It could also serve the prevention objective, since the charge could be directly proportional to the number of employees exposed and the extent of their exposure. In this form, an exposure charge could support the implementation of the ALARA principle (As Low As Reasonably Achievable), to which we attach great importance. (See Chapter 8.) It could become an additional incentive for achieving the lowest practical level of exposure because the amount paid would decrease as measured exposure below the control limit fell. At this juncture, the financing of workers' compensation through exposure charges would have become a useful adjunct in the implementation of the designated substances regulations of the Ministry of Labour. Furthermore, the Advisory Council on Industrial Disease Policy, whose creation we have prescribed, would be the logical body to develop exposure charge policies, substance-by-substance.

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[95] Ibid., pp. 131–134.

If we are ourselves attracted by the merits of the exposure charge idea in principle, we must also acknowledge that the asbestos experience underlines genuine practical limitations. A formidable difficulty with exposure charges can be determining the charge rate: How many dollars per employee per year must the employer pay? The premium cannot be based upon the resulting disease, because that disease will not manifest itself, by definition for long-latency diseases, until decades after the exposure. It might be based upon current disease claims being paid by the Workers' Compensation Board. This has the disadvantage, however, that current disease is the outcome of past rather than present exposure and may not have originated with present employers.

Alternatively, the exposure charge could be based upon forecasts of future disease derived from epidemiological studies. An exposure charge is most likely to be levied upon substances whose hazard has been well-established. It might in fact be limited only to designated substances. By the time a substance is designated, it often has been used for many years, and its hazards may be well known. In the case of asbestos, numerous epidemiological studies allowed us to make a number of forecasts of future disease rates, as shown in Chapter 7. With respect to workplace hazards as thoroughly studied as asbestos, the data base exists for basing the charge upon forecasts of disease.

That very data base, however, can be used to regulate exposure levels or to ban a hazardous substance from the workplace altogether. We ourselves use the data base for these purposes in Chapter 7. In the result, we either restrict or ban many industrial applications of asbestos; the principal activity we condone in fixed place industry is chrysotile manufacturing at a control limit of 1 fibre per cubic centimetre (f/cc), textile manufacturing excepted. It remains possible to calculate an exposure charge from a forecast of possible disease in this regulated activity, but the utility of the exposure charge, as a practical matter, becomes questionable because its level is trivial. As we point out in Chapter 7, an enforced control limit of 1 f/cc means a true average worker exposure of 0.5 f/cc. The volume of disease that might materialize at this level is sufficiently low that a brake manufacturing employer in 1983 would, if presented with an exposure charge calculated on the basis of the present value of future claims from forecast disease, pay less than $10 per year per exposed employee. This amount is inconsequential in comparison to the cost of reducing employee exposure further, which we estimate to be in the thousands of dollars per employee. (See Chapter 6.) It follows that in this instance, an exposure charge generates no incentive for lower exposure.

While this hard case deflates the full-blown argument that can be made for exposure charges in principle, it does not in our view obviate the notion of exposure charges altogether. There are instances where exposure charges might be based on arbitrary figures rather than present disease claims or forecasts of future disease. More likely than not, however, exposure charges will find their potential with substances other than asbestos. Instances where such charges might be developed by the Board should be identified with the co-operation of the Ministry of Labour and the assistance of the Advisory Council on Industrial Disease Policy. We recommend that:

14.11 The Workers' Compensation Board should be sensitive to the potential of exposure charges on a selective basis, but should take notice that asbestos is likely not appropriate for such charges. Instances where such charges might appropriately be developed should be identified with the co-operation of the Ministry of Labour and the assistance of the Advisory Council on Industrial Disease Policy.

Part VII

Learning from the Asbestos Experience

Chapter 15 Identifying and Designating Hazardous Substances

A. Introduction

To learn from the asbestos experience is to learn from the harshest of schoolmasters — human tragedy. It is a fact that society has all too often required this stern tutelage to assimilate its lessons. This is strikingly evident in the realm of regulation. One can think of the Titanic and its impact on the regulation of radio communications at sea, of typhoid epidemics and their impact on the regulation of drinking water, of mine cave-ins and their impact on the regulation of mining. One can think of asbestos. This entire Report has been an exercise in learning from the asbestos experience and discerning the lessons it teaches about health hazards and their regulation. In addition to the many lessons addressed so far, we consider that the asbestos experience teaches lessons on two other subjects: the identification of hazardous substances; and the designation of substances that occasion health hazards in the workplace. We address these topics in this, the final chapter of our Report.

B. Hazard Identification

B.1 Occupational Exposure

It is now universally recognized that the exposure of workers to asbestos has left an appalling legacy of human disease and death. Estimates we canvass in Chapter 14 indicate that between 100,000 and 200,000 workers in the United States will ultimately die from workplace exposures to asbestos. Most of these U.S. workers were exposed to asbestos beginning in the 1940s. Yet a report to the British Parliament in 1930 stated "... that the inhalation of asbestos dust over a period of years results in the development of a serious type of fibrosis of the lungs."[1] This report led to the adoption in 1931 of regulations on the exposure of workers to asbestos in the United Kingdom.[2] How could so many workers be exposed to ultimately fatal asbestos dust levels after an official study had determined that asbestos posed a health hazard and the substance had come under regulation?

The answer is in part that while the British study linked asbestos exposure with worker disease, it did not indicate the relationship between exposure levels and the volume of disease, nor did it identify all relevant diseases. This 1930 report dealt only with asbestosis; the linkage between asbestos and lung cancer was not suggested until the mid-1930s,[3] was not systematically demonstrated until Doll published his seminal work in 1955,[4] and was not widely appreciated until the Selikoff study was published in 1964.[5] Mesothelioma was not clearly linked to asbestos exposure until the work of Wagner, published between 1960 and 1965.[6]

Furthermore, while a study might link asbestosis with historical asbestos exposures, it was possible to argue that if exposure were visibly reduced, the disease might be greatly reduced or eliminated. Thus, Lanza, referring in 1936 to the United States and England, could state that ". . . energetic steps have been taken to control the dust hazard in asbestos plants, so that it is probable that further cases of disabling asbestosis will be rare. . . ."[7] In 1938, the seriously flawed Dreessen study supported the linkage between asbestos exposure and asbestosis, but managed to conclude that ". . . if the dust concentration in asbestos factories could be kept below 5 million particles [per cubic foot]. . . new cases of asbestosis probably would not appear."[8] Thirty years later, in 1968, the Committee on Hygiene Standards of the British Occupational Hygiene Society made a similar conclusion, but the associated level of exposure was 2 fibres per cubic centimetre (f/cc), a tiny fraction of 5 million particles per cubic foot (mppcf).[9] This 1968 finding was itself disputable; even more significantly, however, lung cancer and mesothelioma apparently were not major considerations in establishing the 2 f/cc recommendation. The differing risks associated with crocidolite and amosite as compared to chrysotile in causing mesothelioma were not reflected in regulations until the 1970s and even today are not universally accepted, as evidenced in the United States by the Occupational Safety and Health Administration's (OSHA) 2 fibre per cubic centimetre standard for all types of asbestos, and by OSHA's November 1983 Emergency Temporary Standard of 0.5 f/cc, also for all types of asbestos (subsequently stayed by court order). The widely publicized 1976 National Institute for Occupational Safety and Health (NIOSH) recommendation that all workplace exposures in the United States should be controlled to a limit of 0.1 f/cc is likewise blind to the differing risks from different fibre types. As for the different risks posed by the use of asbestos in different industrial processes, we know of no jurisdiction in the world that regulates asbestos accordingly. To the best of our knowledge, ours is the first official study to frame formal recommendations to the effect that asbestos requires different regulatory responses in different industrial processes.

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[1] E.R.A. Merewether and C.W. Price, *Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry* (London: His Majesty's Stationery Office, 1930).

[2] U.K., Asbestos Industry Regulations (1931), Statutory Rules and Orders, 1931, no. 1140 (London: His Majesty's Stationery Office, 1931).

[3] K.M. Lynch and W.A. Smith, "Pulmonary Asbestosis III. Carcinoma of Lung in Asbesto-silicosis," *American Journal of Cancer* 14 (1935): 56–64; S.R. Gloyne, "Two Cases of Squamous Carcinoma of the Lung Occurring in Asbestosis," *Tubercle* 17 (October 1935): 5–10; S.R. Gloyne, "A Case of Oat Cell Carcinoma of the Lung Occurring in Asbestosis," *Tubercle* 18 (December 1936): 100–101; and D.S. Egbert and A.J. Geiger, "Pulmonary Asbestosis and Carcinoma, Report of a Case with Necropsy Findings," *American Review of Tuberculosis* 34 (July 1936): 143–146.

[4] Richard Doll, "Mortality from Lung Cancer in Asbestos Workers," *British Journal of Industrial Medicine* 12 (1955): 81–86.

[5] Irving J. Selikoff, Jacob Churg, and E. Cuyler Hammond, "Asbestos Exposure and Neoplasia," *Journal of the American Medical Association* 188 (1964): 22–26.

[6] J.C. Wagner, C.A. Sleggs, and P. Marchand, "Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province," *British Journal of Industrial Medicine* 17 (1960): 260–271; and J.C. Wagner, "Epidemiology of Diffuse Mesothelial Tumors: Evidence of an Association from Studies in South Africa and the United Kingdom," *Annals of the New York Academy of Sciences* 132, Art. 1 (31 December 1965): 575–578.

[7] A.J. Lanza, "Asbestosis," *Journal of the American Medical Association* 106 (1 February 1936): 368–369.

This chronicle of events demonstrates that the process of hazard identification can unravel slowly, and that regulatory responses can lag behind the knowledge that slowly accumulates while a mounting toll of disease and death is borne by workers who are thereby cast in the role of human guinea pigs. Is there no alternative? There can be no iron-clad assurance that the asbestos experience will not be repeated. A hazard may initially be identified in relation to one disease when it is in fact associated with others as well. Different types of a single substance may pose different risks. Then there is the matter of dose-response or the extent to which different levels of exposure give rise to different rates of disease. Such uncertainties cannot be wished away and mean that a repetition of the asbestos experience is always in the realm of the possible.

Surely, however, there is an alternative that lies in swifter identification through animal experiments and other techniques and swifter regulatory response. And the good news is that there has been a transformation in society's perception of health and the factors influencing health.

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[8] Waldemar C. Dreessen et al., *A Study of Asbestosis in the Asbestos Textile Industry*, U.S. Public Health Bulletin, no. 241 (Washington, D.C.: U.S. Government Printing Office, August 1938), p. 117.

[9] British Occupational Hygiene Society, Committee on Hygiene Standards, "Hygiene Standards for Chrysotile Asbestos Dust," *Annals of Occupational Hygiene* 11 (1968): 47–69.

In the last forty years, our improved knowledge of the principal factors that contribute to life expectancy has led to a much clearer appreciation of the effects of occupation, environment, nutrition, and lifestyle. The development of our understanding of cancer, its causes, and the limited value of medical therapy has led some distinguished scientists to emphasize that the eventual control of cancer does not reside in cure but in prevention.[10] With the great increase in life expectancy that occurred in this century as a result of the prevention of premature death from infectious disease, our attention is now focused on diseases such as cancer that cause premature death in the adult population. It was not until the 1950s that the recognition of the relationship between smoking and lung cancer began to be established. In the workplace, we have become sensitized to the importance of disease prevention because of the death occasioned by past use of hazardous substances. Society now recognizes the important contribution of factors in the human environment to premature death, that medical treatment is limited in what it can do for many diseases, and that for these diseases prevention is more effective than cure. Society's appreciation of these facts is spawning more effective approaches to hazard identification and swifter regulatory response. Interestingly, there is a facet of the asbestos experience which simultaneously testifies to society's new appreciation of the importance of disease prevention and to the new swiftness of regulatory response. This facet of the asbestos experience, however, involves another lesson: the importance of being discriminating in the formulation of regulatory responses.

B.2 Non-Occupational Exposure

As we have seen, the process of identifying asbestos as an occupational hazard has unravelled slowly, as have the strictness and sophistication of the regulatory response. In the non-occupational realm, the story is very different. By the 1970s, society was sensitized to the contribution of the environment to disease and to the importance of prevention. As the enormity of the public health disaster that had befallen asbestos workers came to be appreciated by the public during the 1970s, regulations were imposed and protective actions were taken in situations which, given today's information, represent understandable but unwarranted over-reaction to public concern about what had been identified as a dangerous workplace killer. Beginning in the mid-1970s, school systems across the United States and Canada launched massive efforts to remove asbestos from school buildings because of parental concern over health effects on their children. In his study for this Commission, Professor G. Bruce Doern presented a history of the treatment of asbestos in schools in Ontario, noting that in 1980 the death of a Scarborough school carpenter from mesothelioma occasioned by occupational exposure heightened public apprehension in Ontario about risks to building occupants.[11] While this was an occupational death, parents reasoned that if a school carpenter could be killed by asbestos in schools, so could school children. This gave impetus to the programme for removing asbestos from schools in Ontario. As we indicate in Chapter 10, there are occasions when asbestos removal is warranted, but these will almost always be justified by the need to reduce the exposure of building workers to asbestos, rather than the need to reduce the exposure of building occupants. Thus, even though the deaths that led to asbestos being identified as a problem in buildings were work-related deaths, asbestos was misapprehended as a hazard to building occupants. We show in Chapter 9 that unless asbestos is being disturbed or falling onto the building surfaces, the health risk it poses to building occupants is insignificant. The exposure of building occupants would generally be 1/10,000, with a few exposures as high as 1/1,000 of those which led to disease among building insulation workers. Yet, throughout Canada and the United States, hundreds of millions of dollars have been spent removing asbestos from buildings in the belief that this would provide protection for building occupants.

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[10] Richard Doll and Richard Peto, "The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today," *Journal of the National Cancer Institute (JNCI)* 66:6 (June 1981): 1191-1308.

How could asbestos in buildings have been misidentified as a public health concern, at such tremendous public expense? Part of the answer lies in the absence, until very recently, of reliable measurements of occupant exposures to asbestos. The special difficulties of measuring airborne asbestos levels in buildings, which are catalogued in Chapter 9, meant that there was little reliable data that could be used to demonstrate the absence of significant airborne fibre levels until recently. Furthermore, the difficulties in using epidemiological studies to detect small increases in the lung cancer rate meant that it was difficult to prove that building exposures were not causing increased disease rates. Yet it remains intriguing that in the workplace context, the absence of information led to under-regulation, while in the buildings context, the absence of information led to over-reaction. This over-reaction was not abated by the U.K. Advisory Committee on Asbestos, which reported in 1979 that:

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[11] G. Bruce Doern, *The Politics of Risk: The Identification of Toxic and Other Hazardous Substances in Canada*, Royal Commission on Asbestos Study Series, no. 4 (Toronto: Royal Commission on Asbestos, 1982), chap. 2.

> . . . unless contaminated buildings are very much commoner than seems likely no appreciable mortality from lung cancer can be associated with any degree of contamination by chrysotile likely to be encountered in the U.K. in the ambient air or in buildings not under active construction or repair. However we emphasise once more the need for further information about asbestos levels in buildings.[12]

The Advisory Committee then recommended a survey to determine dust levels more accurately and concluded that:

> Our evidence of the non-occupational risk is not such as to prompt us to recommend the general removal of asbestos from existing buildings.[13]

The buildings experience demonstrates the critical importance of discriminating among the risks that may be presented by a substance. Something that has been a killer in the workplace may not require control actions in other situations. Other examples of hazard misidentification related to asbestos are documented in this Report. Thus, for instance, the use of asbestos in hairdryers has been regulated despite the absence of any evidence that significant fibre release was possible, while the sale of free-form asbestos is still unregulated.[14] The use of asbestos filters in wine-making has been regulated, despite the absence of evidence that drinking asbestos-containing fluids can present health risks.[15] The asbestos content of drinking water supplies has been the subject of emotional public debate, despite the absence of evidence of any health risk from this source.[16] In the Toronto subway, asbestos brake linings were replaced with non-asbestos brake linings, despite low asbestos fibre levels in the subway, and the brief exposure of the general public to the subway atmosphere.[17]

C. Observations and Recommendations

In his study for this Commission, Professor Doern reflected generally on problems that beset the process of identifying hazardous substances and suggested various approaches that could be taken to improve the process.[18]

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[12] U.K., Advisory Committee on Asbestos, *Asbestos — Volume I: Final Report of the Advisory Committee* (Simpson Report), William J. Simpson, Chairman (London: Her Majesty's Stationery Office, 1979), paragraph 132, p. 63.

[13] Ibid., paragraph 248, p. 91.

[14] See Chapter 11, Section A of this Report.

[15] See Chapter 11, Section B of this Report.

[16] Ibid.

[17] See Chapter 11, Section C of this Report.

[18] Doern, *The Politics of Risk: The Identification of Toxic and Other Hazardous Substances in Canada*, chap. 5.

We commend this study to readers who wish to pursue the subject, and we offer here a distillation of our own observations. Given a hazardous substance that has been studied as thoroughly and for as many years as asbestos, one way to probe the dimensions of the hazard and to discern discriminating regulatory responses is through an inquiry as elaborate as this one. We are content to leave the merits of this approach to the judgement of readers of this Report, and of other similarly elaborate studies, whether of asbestos or other substances, in other jurisdictions. Even if this approach has merit, it is in our view, given its cost in money and time, clearly not a substitute for the development of ongoing, institutionalized approaches to the matter of hazard identification in both the public and private sectors.

During the last decade a number of approaches have been developed to determine whether potentially hazardous substances can be pre-screened in laboratory and animal experiments before humans become exposed to them, and thus avoid unnecessary exposure of humans to damaging substances. There are encouraging signs that the private sector is increasingly promoting these developments, spurred on, at least in part, by the example of the costly litigation that is so clearly devastating the asbestos industry in the United States. Some major chemical companies provide detailed information about the hazardous nature of materials to firms and organizations wishing to use the substance. These companies may even require the firm or organization to which the substance is being sold to provide evidence that they can use the substance safely. This development is most welcome, not least because hazard identification has been often complicated by private sector concern with maintaining trade secrets.

However welcome ongoing developments in the private sector may be, governments clearly need to enhance their own capacities in the realm of hazard identification. Efforts have either been mounted or suggested in this regard, but the picture, at least in Canada and the United States, remains spotty. The U.S. Environmental Protection Agency (EPA) is empowered by the *Toxic Substances Control Act* to require industry to supply information on how and to what extent people and the environment are exposed to certain chemicals, to notify the EPA about new substances to be produced, and to supply the results of studies about the hazards of chemicals.[19] The U.S. Occupational Safety and Health Administration adopted in 1980 "The OSHA Cancer Policy," which classifies suspected carcinogens into two categories depending upon the evidence of carcinogenicity, treats some scientific questions as resolved and not subject to question in the rule-making process, and presents a system of setting priorities in rule-making.[20]

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[19] *Toxic Substances Control Act of 1976*, 15 U.S.C. §2601–2629 (1981).

[20] U.S., Department of Labor, Occupational Safety and Health Administration, "Identification, Classification and Regulation of Potential Occupational Carcinogens," 29 CFR Part 1990, 45 FR 5002–5296, 22 January 1980.

The Policy has never been fully implemented because of court challenges and opposition to a number of its aspects.[21]

The difficulties that "The OSHA Cancer Policy" has experienced aside, we expressed in the course of our hearings our own interest in the extent to which liaison has developed in the United States between EPA and OSHA. Our interest, of course, stemmed from the extent to which asbestos exemplifies a substance that occasions different risks in the workplace and in the environment. Dr. Bailus Walker, Jr., former Director of Health Standards Programs at OSHA, testified before us that he had personally convened a meeting with EPA concerning "The OSHA Cancer Policy," and that there were indeed a number of occasions when the two agencies examined the implications of occupational control for environmental control and vice-versa. However, in Dr. Walker's words, "If there had been some facilitating mandate from Congress or from the Executive Branch, I think [the liaison] would have been far more productive."[22]

The Canadian context of hazard identification differs in many ways from the American, but features the similar absence of a mandate from the highest echelons of government to develop coherence and co-ordination. At the federal level, the Canadian *Environmental Contaminants Act* provides for the identification of the entry of chemicals into the environment and an assessment of their probable impact.[23] Chemicals may be placed in one of three categories depending on the hazard they present. At the provincial level, the Ontario Ministry of the Environment exercises responsibility for the identification and control of hazards under no fewer than four statutes.[24] Meantime, the *Occupational Health and Safety Act* gives the Ontario Ministry of Labour its own mission with respect to regulating workplace hazards and enjoins employers to reveal the nature of substances they introduce in the workplace. The Minister of Labour's Advisory Council on Occupational Health and Occupational Safety has developed a Discussion Paper on the control of workplace carcinogens.[25] There may be liaison between the environmental and occupational realms, for example, through the Ontario Deputy Ministers' Committee on Occupational and Environmental Health, and across federal and provincial jurisdictions. But there remains an absence of structure, an absence of a mandate.

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[21] Doern, *The Politics of Risk: The Identification of Toxic and Other Hazardous Substances in Canada*, p. 4.12.

[22] Ontario, Royal Commission on Asbestos, Transcript of Public Hearings, Evidence of Dr. Bailus Walker, Jr., 20 May 1982, Volume no. 36, p. 123.

[23] *Environmental Contaminants Act*, S.C. 1974-75-76, c. 72.

[24] *Environmental Protection Act*, R.S.O. 1980, c. 141; *Ontario Water Resources Act*, R.S.O. 1980, c. 361; *Pesticides Act*, R.S.O. 1980, c. 376; and *Environmental Assessment Act*, R.S.O. 1980, c. 140.

[25] Ontario, Advisory Council on Occupational Health and Occupational Safety, "The Control of Workplace Carcinogens: A Discussion Paper Prepared by the Advisory Council on Occupational Health and Occupational Safety," Toronto, 30 June 1982. (Mimeographed.)

In 1977, the Science Council of Canada, which is a federal government advisory body on science policy, published a report entitled *Policies and Poisons*. As Professor Doern summarized it:

> The report analyzed the containment of long-term hazards to human health in the environment and in the workplace. . . . To enable Canada to have a better process of risk assessment and a better process for the determination of acceptable risks, the Science Council recommended that a National Advisory Council on Occupational and Environmental Health (NACOEH) be established by statute. NACOEH's mandate would be to designate hazards and ensure that assessments of risks are undertaken and published in respect of any hazard it may designate. It would also be responsible for publishing recommended standards of maximum permissable exposure levels for Canada. NACOEH would evaluate and recommend which hazards should be subjected to regulatory control but it would *not* be the regulator.[26]

After six years, the recommendation of the Science Council still awaits a federal response. While the federal government did involve itself in the creation of an organization called the Canadian Centre for Occupational Health and Safety, this body, in Professor Doern's words, ". . . can in no way be considered an aggressive new initiative in occupational health and safety nor is it a research agency such as that envisaged by the Science Council. . . ."[27] We have no comment on whether the precise structure recommended by the Science Council is more appropriate than other alternatives, but we urgently invite governmental response to the need for a national hazard identification agency whose mandate involves identification and assessment, not regulation, and that transcends the realms of occupational and environmental health. Because research results are a common pool of knowledge useful to all governments, such an agency can most effectively be mounted as a national agency with a capacity to liaise extensively with hazard identification agencies in other countries.

There are three alternative ways such a national agency could come into being. Under the first alternative, the federal government could create a new agency or designate one of its existing agencies, for example, the National Research Council, to fill the role. Such an agency would thereupon develop extensive links with the provinces. In the second alternative, the federal and provincial governments could jointly create and finance an intergovernmental hazard identification agency. The third option would envisage vesting the hazard identification role in a non-governmental institution which could be either university-connected (for example, the Canadian Centre for Toxicology, which is based in the Universities of Guelph and Toronto) or be a free-standing, non-profit institution. Whatever the alternative, we express the view that the Government of Ontario, to which we report as a Commission, should stimulate other Canadian governments to work towards the expeditious establishment of a national hazard identification agency, and we accordingly recommend that:

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[26] Doern, *The Politics of Risk: The Identification of Toxic and Other Hazardous Substances in Canada*, pp. 4.17–4.18.

[27] Ibid., p. 4.19.

15.1 The Government of Ontario should take steps, using its established channels of federal-provincial communication, to promote the expeditious development of a national agency for hazard identification and risk assessment that would serve the needs of governments at all levels of jurisdiction. This agency might be a federal agency, an agency jointly created by the federal government and the provinces, or a university-based centre or free-standing, non-profit institution with a mandate and funding provided jointly by the federal and provincial governments.

A national hazard identification and risk assessment agency should not, and indeed cannot, have a monopoly over these functions. Operating ministries such as the Ministry of Labour and the Ministry of the Environment will, in the normal course of discharging their regulatory responsibilities, uncover information that leads them to identify hazards of various types. A national agency can be in a position to provide valuable information, but it cannot assume all the hazard identification and risk assessment tasks that are associated with ongoing regulatory responsibilities.

The need for communication among operating ministries with such responsibility remains. Equally acute is the need for co-ordinated communication between these ministries, on the one hand, and the public, on the other. Nowhere is this need more tellingly illustrated than in the development of standards and guidelines for asbestos exposure in workplaces and in buildings. The Ontario Ministry of Labour has a control limit of 1 f/cc for chrysotile exposure in workplaces. Meantime, the Ontario Ministry of the Environment has a guideline of 0.04 f/cc for asbestos in the ambient air generally extrapolated to building air. A crucial feature of these two reference numbers is that they cannot be directly compared, because they are based on different measurement techniques.[28] The transmission electron microscope (TEM) used for ambient air quality monitoring will detect and report fibres thinner than can be seen and counted using the optical microscope (PCM) that is routinely applied for workplace monitoring. Thus, as we see in Chapter 9, a TEM environmental fibre count will in general be overstated in comparison to a PCM fibre count by perhaps one order of magnitude, that is, approximately 10 times. It follows that the Environment guideline is less like 1/25 and more like 1/250 the Labour control limit for chrysotile exposure. This appears to us not to be recognized by most officials in these agencies; even less has it been communicated to the public. There is thus rampant confusion about the interpretation of air quality measurements made in buildings and unwarranted alarm raised among building occupants who have not been made aware of the differences that must govern the interpretation of TEM and PCM measurements. We consider that the Ministries of Labour and of the Environment should jointly seize the opportunity, as soon as possible after the release of this Report, to prepare an information sheet which, in the simplest possible terms, would explain to the public the differences between TEM and PCM counts and the consequent irrelevance of making a direct comparison between the numerical Labour control limits and the numerical Environment guideline. We therefore recommend that:

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[28] See Chapter 11, Section C.1 of this Report.

15.2 The Ministries of Labour and of the Environment should, as soon as possible after the release of this Report, prepare and disseminate an information sheet which explains, in the simplest possible terms, the differences between the measurement techniques used to derive fibre counts in fixed workplaces and the measurement techniques used to derive fibre counts in building air, and of the consequent irrelevance of making direct comparisons between the numerical asbestos control limits of the Ministry of Labour and the numerical guidelines of the Ministry of the Environment.

We have a final observation in the realm of hazard identification and public communication which pertains to the role of the media and of politicians. While we have argued in favour of better co-ordination among government departments, and for a hazard identification and risk assessment agency that will cut across departmental and federal-provincial lines, we firmly believe that such "bureaucratic solutions" are not a substitute for the role that the media and politicians have played and will play. The charge that media and politicians have sometimes over-dramatized hazardous substances is not without foundation, but neither is the charge that bureaucratic solutions are the products of neat and tidy minds whose objective is to permit governments to enjoy the quiet life. Such charges all too easily obscure what is desirable and indeed invaluable. The media have a crucial role in bringing hazards to the attention of the public, both through independent investigative journalism and as a forum for publicizing scientific findings. Elected representatives, not least as recipients of information from their constituents, can sometimes be uniquely situated to bring hazards to the attention of governments and the public. More generally, in our system of responsible government, they act as the agents of the electorate to test the efficacy of hazard identification and regulation. To the extent that over-dramatization by the media and politicians, or bureaucratic pursuit of the quiet life, can sometimes prevail, the issue should be faced and not circumvented. To this end, a national hazard identification and risk assessment agency should encourage open contact among journalists, politicians, and public servants.

D. The Process of Designating Hazardous Substances

During the existence of this Commission, asbestos became a designated substance under the *Occupational Health and Safety Act*.[29] Section 41(1) of that Act authorizes the Lieutenant Governor in Council to make regulations, and section 41(2), paragraph 14 specifically authorizes "prescribing any biological, chemical or physical agent or combination thereof as a designated substance," while section 41(2), paragraph 15 authorizes "prohibiting, regulating, restricting, limiting or controlling the handling of, exposure to, or the use and disposal of any designated substance." Section 22 of the Act stipulates that before a substance can be designated under section 41, the Minister is to give public notice of intent and call for briefs or submissions in relation to the designation. Pursuant to the Act, the Ministry of Labour has established a process for designating hazardous substances. This process begins with the notice of intent to designate a substance, which, in the case of asbestos, was issued on June 28, 1980.[30] On August 16, 1980, a draft regulation on asbestos was published.[31] During the latter part of 1980 and the first half of 1981, 54 briefs were received from interested parties regarding the proposed asbestos regulation. In September of 1981, a revised regulation was issued by the Ministry of Labour, responding in part to the briefs that had been received to that point.[32] The proposed regulation was subsequently reviewed by the Minister's Advisory Council on Occupational Health and Occupational Safety during the spring of 1982. Finally, on August 20, 1982, the Regulation Respecting Asbestos was filed.[33]

While the process just described is commendable in the degree of public consultation that it involves, it is evidently slow. More than two years elapsed between the formal announcement that asbestos was to be designated and the issuance of the resulting regulation. While the designation of other substances has proceeded somewhat more rapidly, the welcome requirements of openness and submissions mean that the process is inherently time-consuming.

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[29] R.S.O. 1980, c. 321.

[30] Ontario, Ministry of Labour, "Notice of Intention," *The Ontario Gazette*, vol. 113-26, 28 June 1980, p. 2641.

[31] Ontario, Ministry of Labour, "Proposed Regulation under *The Occupational Health and Safety Act, 1978*: Asbestos — Designated Substance," *The Ontario Gazette*, vol. 113-33, 16 August 1980, pp. 3339-3348.

[32] Ontario, Ministry of Labour, Occupational Health and Safety Division, "Proposed Regulation under *The Occupational Health and Safety Act, 1978* and Related Codes — Asbestos — Designated Substance," 22 September 1981. (Mimeographed.)

[33] Regulation Respecting Asbestos, O. Reg. 570/82, made under the *Occupational Health and Safety Act*, R.S.O. 1980, c. 321.

The notice of intent to designate indicates the Ministry's belief that a substance poses a significant hazard to the health of workers. The issuance of a regulation for a designated substance presumably enhances significantly the protection from that substance offered to workers. During the long period between issuance of a notice to designate and the emergence of the final regulation, workers continue to be exposed to a known hazard, without additional regulatory protection. In the case of asbestos, there was no prior control limit, but instead a guideline of 2 f/cc for occupational asbestos exposure except that a 0.2 f/cc guideline applied to crocidolite.[34] In workplaces with less than twenty employees, no joint health and safety committee was required, although this was required once the Regulation came into effect. And while a number of protective measures were in place, they lacked a status sufficient to make their violation a clear-cut legal offence.[35]

The absence of direct regulations while a substance is in the process of designation might be a matter of little concern if the process required only a few months. Where the process may require two years, the regulatory regime during this period is of considerable importance. It would obviously be desirable to impose such protection as could be provided during this interim period while waiting for the final regulation to be issued. This might be achieved by issuing a preliminary regulation as soon as possible after the notice of intent to designate is given. This preliminary regulation would protect workers while the final regulation was in the process of development. The preliminary regulation could specify those elements of the anticipated final regulation that were not seriously in controversy at the time. For example, if a guideline for worker exposure was in effect, that guideline might be adopted as a control limit by the preliminary regulation. If standardized measurement methods and respiratory protection were available, they too could be incorporated in the preliminary regulation, even though they might be altered in the final regulation. A joint health and safety committee could be required in all workplaces where the substance was present. The employer could be required to develop a control programme and a monitoring programme, in collaboration with the joint health and safety committee. The scope of the preliminary regulation might vary from one substance to another, depending upon what pre-existing standards, procedures, and methods were sufficiently accepted to constitute interim regulation. In the case of asbestos, all of the items listed above could have been made effective at the date when the notice of intent to designate was made.

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[34] See Chapter 3, Section B of this Report.

[35] *Her Majesty the Queen v. The Windsor Board of Education and Eric Laub* in the Provincial Court (Criminal Division), County of Essex, His Honour Judge S. Nosanchuk, 11 June 1982, unreported.

It is possible that section 20 of the *Occupational Health and Safety Act* might provide a basis for the interim protection of workers during the designation of a substance. Section 20(1)(c) allows a Director within the Ministry to issue to employers orders which can specify ". . . administrative control, work practices, engineering control and time limits for compliance. . . ." Section 20, however, applies to an individual employer, so that the Director would be required to issue orders individually to each employer thought to be using the hazardous substance. Furthermore, section 20 requires reasons and opinions of the Director of the branch, and each order can be appealed to the Minister. The result of this is that section 20 has rarely been used, and it seems not to be a strong basis for interim regulation of hazardous substances.

A preferable alternative would be for the Ministry simply to proceed in two stages under section 41(2), paragraphs 14 and 15. After the notice of intent to designate was issued, the Ministry could formally designate the substance and adopt the preliminary regulation as soon as a preliminary regulation was ready. The process of developing the final regulation would continue, and when a final regulation was approved, it could replace the preliminary regulation. Given the notice requirements of section 22, however, it is likely that the Act would have to be amended to permit such a two-stage process.

The asbestos experience speaks for change which will afford an enhanced measure of worker protection during the time consumed by the process of substance designation. However, because asbestos has now been designated and other substances, which fall outside our terms of reference, are involved, we refrain from making a direct and detailed recommendation. The Minister's Advisory Council on Occupational Health and Occupational Safety is a representative body that includes members of labour and management. In our view, it is ideally situated to advise on the changes which, while maintaining the openness and thoroughness of the designation process, would permit the introduction and enforcement of interim regulations while this process unfolds. In this light, we recommend that:

15.3 The Minister of Labour should make a formal reference to the Advisory Council on Occupational Health and Occupational Safety concerning what measures, including statutory amendment if necessary, should be taken to permit the preliminary regulation of designated substances while the designated substance process is underway.

We consider that a two-stage procedure for designated substances would have several benefits. First and foremost, it would provide workers with some additional protection during the long process of designation.

Second, it would provide the Ministry of Labour with the legal basis for requiring those employers who might not voluntarily comply with a draft regulation to follow the good example set by those who do. It also preserves horizontal equity among employers in ensuring that all are following the same rules during the designation process.

Appendices

Appendix A

Parties Granted Legal Standing by the Royal Commission on Asbestos

Party	Liaison Person(s)	Representative(s) at Hearings
1. Asbestos Information Association/North America	Mr. Timothy S. Hardy	Mr. Timothy S. Hardy Mr. Arthur F. Sampson III Mr. Edward W. Warren
2. Asbestos Victims of Ontario	Mr. Ed Cauchi Mr. David K.L. Starkman	Mr. Ed Cauchi Mr. David K.L. Starkman
3. Canadian Union of Public Employees, Ontario Division	Mr. Jack Bird	Through the Ontario Federation of Labour (OFL)
4. Communications Workers of Canada, Ontario Region	Mr. Gary Cwitco	Through the OFL
5. Energy and Chemical Workers Union	Mr. Robert Stewart Mr. Daniel Ublansky	Mr. Daniel Ublansky Also, through the OFL
6. Government of Ontario	Mr. Arthur L. Gladstone	Mr. Thomas R. Lederer Mr. James McNamee
7. Injured Workers' Consultants	Mr. Nick McCombie	Mr. Orlando Buonastella Mr. Nick McCombie
8. International Association of Bridge, Structural and Ornamental Iron Workers, Local 721	Mr. John Donaldson	Mr. David K.L. Starkman
9. International Brotherhood of Electrical Workers, Local Union 105	Mr. J. Barry Fraser	Through the OFL

Appendix A (continued)

Party	Liaison Person(s)	Representative(s) at Hearings
10. International Union, United Automobile, Aerospace and Agricultural Implement Workers of America, Canadian Region	Mr. James Gill	Through the OFL
11. Johns-Manville Canada Inc.	Mr. Thomas S. Patterson	Mr. Rick Evans
12. Labour Council of Metropolitan Toronto	Mr. Wally Majesky	Through the OFL
13. London and District Labour Council	Ms. Gail Sneddon	Through the OFL
14. Ontario Federation of Labour	Ms. Linda Jolley	Ms. Linda Jolley
15. Quebec Asbestos Mining Association	M^e Phillipe Casgrain	M^e Jean Bazin M^e Phillipe Casgrain
16. St. Thomas and District Labour Council	Mr. Howard Hunter	Through the OFL
17. Toronto Occupational Health Resource Committee	Mr. Andrew King	Dr. Brian Gibson
18. United Electrical, Radio and Machine Workers of America, National Office	Mr. Val Bjarnason	Through the OFL
19. United Steelworkers of America, National Office and District 6	Mr. Lorne Heard Mr. Paul Falkowski Mr. Ken Valentine	Through the OFL
20. Windsor Occupational Safety and Health Council	Mr. James Brophy Mr. John Jackson	

Appendix B

Witnesses Before the Royal Commission on Asbestos

Date	Witness*	Institutional Affiliation**	Transcript Volume Number
11 June 1981	Dr. Philip E. Enterline	Professor and Chairman Department of Biostatistics University of Pittsburgh Pittsburgh, Pennsylvania, U.S.A.	8
16 June 1981	Dr. Hans Weill	Head, Pulmonary Diseases Section Department of Medicine School of Medicine Tulane University New Orleans, Louisiana, U.S.A.	9
18 June 1981 19 June 1981	Mr. Geoffrey Berry	Statistician Medical Research Council Pneumoconiosis Unit Llandough Hospital Penarth, Glamorgan, Wales	10 11

Notes: *Witness called by the Royal Commission on Asbestos, unless otherwise indicated in parentheses.

**Institutional affiliations given for witnesses are those at the time of their testimony. Where the word ''formerly'' is added, it is because the institutional affiliation given is relevant to the work of the Commission.

Date	Witness	Institutional Affiliation	Transcript Volume Number
24 June 1981 25 June 1981	Dr. J. Corbett McDonald	Professor, School of Occupational Health McGill University Montreal, Quebec, Canada; and formerly, Director, TUC Centenary Institute of Occupational Health London School of Hygiene and Tropical Medicine London, England	12 13
29 June 1981 30 June 1981	Dr. William J. Nicholson	Associate Professor, Community Medicine, and Associate Director, Environmental Sciences Laboratory Department of Community Medicine Mount Sinai School of Medicine The City University of New York, New York, New York, U.S.A.	14 15
7 July 1981	Dr. Henry A. Anderson	Chief, Section of Environmental Epidemiology Wisconsin State Division of Health Madison, Wisconsin, U.S.A.; and formerly, Assistant Professor Environmental Sciences Laboratory Department of Community Medicine Mount Sinai School of Medicine The City University of New York New York, New York, U.S.A.	16
8 July 1981	Mr. Richard A. Lemen	Director, Division of Criteria Documentation and Standards Development; and Director, Division of Technical Services National Institute for Occupational Safety and Health Rockville, Maryland, U.S.A.	17

9 July 1981	Dr. Eric J. Chatfield	Head, Electron Optical Laboratory Department of Applied Physics Ontario Research Foundation Sheridan Park Research Community Mississauga, Ontario, Canada	18
20 July 1981	Dr. E. Donald Acheson	Director and Professor, Clinical Epidemiology Medical Research Council Environmental Epidemiology Unit Southampton General Hospital Southampton, England; and Co-author, "The Ill Effects of Asbestos on Health," a report to the U.K. Advisory Committee on Asbestos.	19
21 July 1981	Dr. Margaret R. Becklake	Professor of Epidemiology Department of Epidemiology and Health McGill University Montreal, Quebec, Canada	20
22 July 1981 23 July 1981	Dr. Paul Kotin (Asbestos Information Association/North America)	Senior Vice-President Health, Safety and Environment Johns-Manville Corporation Denver, Colorado, U.S.A.; and formerly, Vice-President for Health Sciences Temple University Health Sciences Center Philadelphia, Pennsylvania, U.S.A.	21(A) 21(B)
24 July 1981	M. Marc Trudeau	Chef, Service d'hygiène du travail Direction des programmes et normes Commission de la santé et de la sécurité du travail du Québec Montréal, Québec, Canada	22

Date	Witness	Institutional Affiliation	Transcript Volume Number
27 July 1981	Dr. Edwin C. Holstein	Assistant Professor Environmental Sciences Laboratory Department of Community Medicine Mount Sinai School of Medicine The City University of New York New York, New York, U.S.A.	23
28 July 1981	Dr. Murray M. Finkelstein	Medical Consultant Special Studies and Services Branch Occupational Health and Safety Division Ontario Ministry of Labour Toronto, Ontario, Canada	24
29 July 1981 30 July 1981	Mr. Julian Peto	Statistician Cancer Epidemiology and Clinical Trials Unit Imperial Cancer Research Fund University of Oxford Oxford, England	25(A) 25(B)
13 August 1981	Dr. Kenny S. Crump (Asbestos Information Association/ North America)	President Science Research Systems, Inc. Ruston, Louisiana, U.S.A.	26
14 August 1981	Dr. Gerald R. Chase and	Biostatistician/Epidemiologist Johns-Manville Corporation Denver, Colorado, U.S.A.	27
	Dr. Harrison B. Rhodes (Johns-Manville Corporation; and Asbestos Information Association/North America)	Manager, Occupational Health Union Carbide Corporation; and Chairman, Air-Monitoring Committee Asbestos Information Association/North America Washington, D.C., U.S.A.	

20 August 1981	Dr. Jacques Dunnigan (Quebec Asbestos Mining Association)	Directeur, Institut de recherche et de développement sur l'amiante Université de Sherbrooke Sherbrooke, Québec, Canada	28
21 August 1981	Dr. Graham W. Gibbs	Director, Health and Safety Affairs Celanese Canada Inc., Montreal, Quebec, Canada; and formerly, Director, Institute of Occupational Health and Safety McGill University Mont St-Hilaire, Quebec, Canada	29
26 August 1981	Dr. Murray M. Finkelstein	Medical Consultant Special Studies and Services Branch Occupational Health and Safety Division Ontario Ministry of Labour Toronto, Ontario, Canada	30
27 August 1981	Dr. Alison D. McDonald	Directeur de programme au département de recherche interne Institut de recherche en santé et en sécurité du travail du Québec Montréal, Québec, Canada; and formerly, Professor and Head Department of Epidemiology St. Mary's Hospital Medical School London, England	31
23 October 1981	Dr. John M. Dement	Safety and Health Manager National Institute of Environmental Health Sciences Research Triangle Park, North Carolina, U.S.A.	32

Date	Witness	Institutional Affiliation	Transcript Volume Number
13 November 1981	Dr. Graham W. Gibbs	Director, Health and Safety Affairs Celanese Canada Inc., Montreal, Quebec, Canada; and formerly, Director, Institute of Occupational Health and Safety McGill University Mont St-Hilaire, Quebec, Canada	33
15 January 1982	Dr. John M.G. Davis	Head, Pathology Branch Institute of Occupational Medicine Edinburgh, Scotland	34
18 February 1982 19 February 1982	Mr. James F. Reis	President Asbestos Information Association/North America Washington, D.C., U.S.A.; and Director, Asbestos Policy Johns-Manville Corporation Denver, Colorado, U.S.A.	35(A) 35(B)
20 May 1982	Dr. Bailus Walker, Jr.	Director, Michigan Department of Public Health Lansing, Michigan, U.S.A.; and formerly, Director, Health Standards Programs Occupational Safety and Health Administration U.S. Department of Labor Washington, D.C., U.S.A.	36
28 May 1982	Mr. Gunnar Danielson and	Director General National Board for Occupational Health and Safety Stockholm, Sweden	37

	Mr. Arne Westlin	Head, Supervision Department National Board for Occupational Health and Safety Stockholm, Sweden	
	and		
	Mr. Lars Ettarp	Director, Department for Working Environment Ministry of Labour Stockholm, Sweden	
	and		
	Mr. Harald Linton	Section Head, Department for Working Environment Ministry of Labour Stockholm, Sweden	
1 June 1982	Mr. William J. Simpson	Chairman, U.K. Health and Safety Commission; and formerly, Chairman, U.K. Advisory Committee on Asbestos London, England	38
8 June 1982	Dr. Jerome J. Vingilis	Member, Advisory Committee on Occupational Chest Diseases Ontario Workers' Compensation Board Toronto, Ontario, Canada; and formerly, Chief, Occupational Chest Disease Service Occupational Health Branch Occupational Health and Safety Division Ontario Ministry of Labour Toronto, Ontario, Canada	39
15 June 1982	Mr. Robert Sass	Associate Deputy Minister of Labour, and Director, Occupational Health and Safety Branch Saskatchewan Department of Labour Regina, Saskatchewan, Canada	40(A)

Date	Witness	Institutional Affiliation	Transcript Volume Number
15 June 1982	Mr. Robert Wilson	Director, Health and Safety Division Ontario Hydro Toronto, Ontario, Canada	40(B)
16 June 1982	Mr. Ross Hunt	Research Physiologist, Industrial Health Research Unit BBA Group PLC Cleckheaton, England	41
21 June 1982	Mr. Gyan S. Rajhans	Chief, Occupational Health Hygiene Service Occupational Health Branch Occupational Health and Safety Division Ontario Ministry of Labour Toronto, Ontario, Canada	42(A)
21 June 1982	Dr. Peter L. Pelmear	Director, Occupational Health Branch Occupational Health and Safety Division Ontario Ministry of Labour Toronto, Ontario, Canada	42(B)
22 June 1982	Mr. Walter Melinyshyn	Director, Industrial Health and Safety Branch Occupational Health and Safety Division Ontario Ministry of Labour Toronto, Ontario, Canada	43(A)
22 June 1982	Mr. James McNair	Special Advisor, Sectoral Studies Occupational Health and Safety Division Ontario Ministry of Labour Toronto, Ontario, Canada	43(B)

23 June 1982	Mr. Jack P. Cashman	President and General Manager Manville Canada Inc. Toronto, Ontario, Canada	44
28 June 1982	Mr. Bruce C. Machin	Vice-President and General Production Manager Manville Canada Inc. Toronto, Ontario, Canada	45(A)
28 June 1982	Mr. Eugene Girdauskas (Injured Workers' Consultants)	Chief Steward Canadian Union of Operating Engineers and General Workers, Local 110 Toronto, Ontario, Canada	45(B)
29 June 1982	Dr. Paul Kotin (Johns-Manville Corporation)	Formerly, Senior Vice-President Health, Safety and Environment Johns-Manville Corporation Denver, Colorado, U.S.A.	46
30 June 1982	Mr. Arthur L. Gladstone	Senior Policy Advisor Occupational Health and Safety Division Ontario Ministry of Labour Toronto, Ontario, Canada	47(A)
13 July 1982	Mr. John F. McDonald	Executive Director Claims Services Division Ontario Workers' Compensation Board Toronto, Ontario, Canada	48
14 July 1982	Dr. Alexander C. Ritchie	Professor of Pathology University of Toronto Toronto, Ontario, Canada	49

Date	Witness	Institutional Affiliation	Transcript Volume Number
14 July 1982	Mr. John F. McDonald	Executive Director Claims Services Division Ontario Workers' Compensation Board Toronto, Ontario, Canada	49
15 July 1982	Dr. Charles Stewart	Chest Disease Consultant Ontario Workers' Compensation Board Toronto, Ontario, Canada	50
16 July 1982	Dr. Cameron C. Gray	Consultant, Advisory Committee on Occupational Chest Diseases; and Special Consultant, Occupational Chest Diseases Medical Services Division Ontario Workers' Compensation Board Toronto, Ontario, Canada	51
16 July 1982	Dr. Douglas W. Dyer	Chest Disease Specialist Ontario Workers' Compensation Board Toronto, Ontario, Canada	51
19 July 1982	Mr. William D. Pearce	Rehabilitation Specialist Ontario Workers' Compensation Board Toronto, Ontario, Canada	52
20 July 1982	Dr. William J. McCracken	Executive Director Medical Services Division Ontario Workers' Compensation Board Toronto, Ontario, Canada	53

20 July 1982	Mr. John F. McDonald	Executive Director Claims Services Division Ontario Workers' Compensation Board Toronto, Ontario, Canada	53
10 August 1982	Mr. Edgar G. Stevens	Industrial Hygienist Manville Canada Inc. Toronto, Ontario, Canada	54
10 August 1982	Dr. E. Keith Fitzgerald	Medical Officer of Health Department of Health Borough of Scarborough Scarborough, Ontario, Canada	54
11 August 1982	Mr. Alan G. MacDonald	Vice-Chairman of Administration and General Manager Ontario Workers' Compensation Board Toronto, Ontario, Canada	55
12 August 1982	Mr. William D. Pearce	Rehabilitation Specialist Ontario Workers' Compensation Board Toronto, Ontario, Canada	56
24 August 1982	Professor Peter S. Barth	Professor of Economics University of Connecticut Storrs, Connecticut, U.S.A.; and Author, *Workers' Compensation and Asbestos in Ontario*, Royal Commission on Asbestos, Study no. 2	57

Appendix C

Exhibits Filed During Royal Commission on Asbestos Hearings

Exhibit Number	Date Filed	During Testimony of:	Transcript Volume Number	Exhibit
II-1	11/06/81	Dr. Philip E. Enterline	8	Compendium of 12 Articles by Dr. Philip E. Enterline.
II-2	11/06/81	Dr. Philip E. Enterline	8	Enterline, Philip E. "Proportion of Cancer Due to Exposure to Asbestos." In *Banbury Report 9: Quantification of Occupational Cancer*, pp. 19–33. Edited by Richard Peto and Marvin Schneiderman. [Cold Spring Harbor, New York]: Cold Spring Harbor Laboratory, 1981. [In press version tabled.]
II-3	11/06/81	Dr. Philip E. Enterline	8	Enterline, Philip E. "Epidemiologic Basis for the Asbestos Standard." Paper presented at the Second Annual Symposium on Environmental Epidemiology, Pittsburgh, Pennsylvania, 28 April 1981. (Mimeographed.)
II-4	11/06/81	Dr. Philip E. Enterline	8	Dement, John M.; Harris, Robert L., Jr.; Symons, Michael J.; and Shy, Carl M. "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers." [In press version tabled.] Later published in *Annals of Occupational Hygiene* 26:1–4 (1982): 869–887.

II-5	16/06/81	Dr. Hans Weill	9	Finkelstein, Murray M. "Asbestosis Among Long-Term Employees of an Ontario Asbestos-Cement Factory." Toronto, Ontario Ministry of Labour, February 1981. (Mimeographed.)
II-6	16/06/81	Dr. Hans Weill	9	Finkelstein, Murray M. "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory: A Preliminary Report." Toronto, Ontario Ministry of Labour, February 1981. (Mimeographed.)
II-7	16/06/81	Dr. Hans Weill	9	Compendium of 16 Articles by Dr. Hans Weill.
II-8	16/06/81	Dr. Hans Weill	9	"Relative Risk (Observed/Expected) with 95% Confidence Intervals by Mean Exposure for Each of Five Exposure Categories." (Hard copy of transparency presented by Dr. Hans Weill.)
II-9	16/06/81	Dr. Hans Weill	9	"Prevalence of Small Opacities"; "Mean Reduction in Lung Function"; and "Risk of Respiratory Malignancy." (Hard copy of slide presented by Dr. Hans Weill.)
II-10	16/06/81	Dr. Hans Weill	9	Adaptation of "Dose-Responses for Lung Cancer." In U.K. Advisory Committee on Asbestos. *Asbestos — Volume 2: Final Report of the Advisory Committee* (Simpson Report), William J. Simpson, Chairman, Figure 11, p. 81. London: Her Majesty's Stationery Office, 1979; with additional dose-response curves drawn in for Weill and Dement studies. (Hard copy of transparency presented by Dr. Hans Weill.)

Exhibit Number	Date Filed	During Testimony of:	Transcript Volume Number	Exhibit
II-11	16/06/81	Dr. Hans Weill	9	Adaptation of "Plot of Lung Cancer and Other Non-malignant Respiratory Disease — SMRs by Cumulative Dose, White Males." In Dement et al. "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Textile Workers," Figure 2, p. 10.4-11. Draft. (See Exhibit II-4 above.) With additional plot lines drawn for different conversion factors. (Hard copy of transparency presented by Dr. Hans Weill.)
II-12	16/06/81	Dr. Hans Weill	9	"Prevalence of Small Irregular Opacities." (Hard copy of transparency presented by Dr. Hans Weill.)
II-13	18/06/81	Mr. Geoffrey Berry	10, 11	Compendium of 16 Articles by Mr. Geoffrey Berry.
II-14	19/06/81	Mr. Geoffrey Berry	11	Peto, Julian. "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory." In *Biological Effects of Mineral Fibres*, Vol. 2, pp. 829-836. Edited by J.C. Wagner. IARC Scientific Publications, no. 30. Lyon, France: International Agency for Research on Cancer, 1980.
II-15	19/06/81	Mr. Geoffrey Berry	11	Rossiter, Charles E., and Berry, Geoffrey. "The Interaction of Asbestos Exposure and Smoking on Respiratory Health." *Bull. europ. Physiopath. resp.* 14 (1978): 197-204.

II-16	19/06/81	Mr. Geoffrey Berry	11	Extra References to 7 Articles by Mr. Geoffrey Berry.
II-17	24/06/81	Dr. J. Corbett McDonald	12	Dr. J. Corbett McDonald — Curriculum Vitae and List of Publications.
II-18	24/06/81	Dr. J. Corbett McDonald	12, 13	Compendium of 29 Articles by Dr. J. Corbett McDonald.
II-19	29/06/81	Dr. William J. Nicholson	14, 15	Compendium of 14 Articles by Dr. William J. Nicholson.
II-20	25/06/81	Dr. J. Corbett McDonald	13	Excerpt from Quebec Asbestos Mining Association. Written submission to the Royal Commission on Asbestos, #16, 14 January 1981, pp. 19, 21, 31, 32.
II-21	25/06/81	Dr. J. Corbett McDonald	13	Excerpt from Epstein, Samuel. *The Politics of Cancer*, p. 84. New York: Anchor Books, 1979.
II-22	25/06/81	Dr. J. Corbett McDonald	13	Excerpt from Chatfield, Eric J. "The Problems of Measurement of Asbestos." In Ontario, Royal Commission on Asbestos. *Proceedings of The Royal Commission on Asbestos, Second Public Meeting, Friday, December 12, 1980*, Appendix A, p. 5. Reported by Lydia Dotto. Toronto: Royal Commission on Asbestos, 1981.
II-23	07/07/81	Dr. Henry A. Anderson	16	Compendium of 9 Articles by Dr. Henry A. Anderson.

Exhibit Number	Date Filed	During Testimony of:	Transcript Volume Number	Exhibit
II-24	07/07/81	Dr. Henry A. Anderson	16	U.S. Environmental Protection Agency. Office of Toxic Substances. *Asbestos-Containing Materials in School Buildings: A Guidance Document. Part I*, chap. 7. Washington, D.C.: U.S. Government Printing Office, 1979.
II-24(a)	07/07/81	Dr. Henry A. Anderson	16	"Wisconsin Asbestos Workshop Educational Materials."
II-25	07/07/81	Dr. Henry A. Anderson	16	Hammond, E. Cuyler, and Selikoff, Irving J. "Passive Smoking and Lung Cancer with Comments on Two New Papers." *Environmental Research* 24:2 (April 1981): 444–452.
II-26	08/07/81	Mr. Richard A. Lemen	17	Compendium of 12 Articles for Mr. Richard A. Lemen.
II-27	09/07/81	Dr. Eric J. Chatfield	18	Compendium of 19 Articles by Dr. Eric J. Chatfield.
II-28	20/07/81	Dr. E. Donald Acheson	19	Compendium of 9 Articles by Dr. E. Donald Acheson.
II-29	21/07/81	Dr. Margaret R. Becklake	20	Compendium of 18 Articles by Dr. Margaret R. Becklake.
II-30	22/07/81	Dr. Paul Kotin	21(A), 21(B)	Compendium of 12 Articles by Dr. Paul Kotin.

II-31(a)	22/07/81	Dr. Paul Kotin	21(A)	Morgan, A. "Fiber Dimensions: Their Significance in the Deposition and Clearance of Inhaled Fibrous Dusts." Harwell, Oxfordshire, 1975. (Mimeographed.)
II-31(b)	22/07/81	Dr. Paul Kotin	21(A)	Morgan, A.; Evans, J.C.; and Holmen, A. "Deposition and Clearance of Inhaled Fibrous Minerals in the Rat: Studies Using Radioactive Tracer Techniques." Harwell, Oxfordshire, 1977. Draft.
II-32	22/07/81	Dr. Paul Kotin	21(A)	Brody, Arnold R.; Hill, Lila H.; Adkins, Bernard, Jr.; and O'Connor, Robert W. "Chrysotile Asbestos Inhalation in Rats: Deposition Pattern and Reaction of Alveolar Epithelium and Pulmonary Macrophages." *American Review of Respiratory Disease* 123 (1981): 670-679.
II-33	24/07/81	M. Marc Trudeau	22	Whitwell, F.; Scott, J.; and Grimshaw, M. "Relationship Between Occupations and Asbestos-Fibre Content of the Lungs in Patients with Pleural Mesothelioma, Lung Cancer and Other Diseases." *Thorax* 32 (1977): 377-386.
II-34	24/07/81	M. Marc Trudeau	22	Compendium of 10 Articles for M. Marc Trudeau.
II-35	27/07/81	Dr. Edwin C. Holstein	23	Compendium of 4 Articles by Dr. Edwin C. Holstein.
II-36	28/07/81	Dr. Murray M. Finkelstein	24	Compendium of 9 Articles by Dr. Murray M. Finkelstein.

Exhibit Number	Date Filed	During Testimony of:	Transcript Volume Number	Exhibit
II-37	29/07/81	Mr. Julian Peto	25(A), 25(B)	Compendium of 12 Articles by Mr. Julian Peto.
II-38	13/08/81	Dr. Kenny S. Crump	26	Compendium of 3 Articles by Dr. Kenny S. Crump.
II-39	14/08/81	Dr. Gerald R. Chase and Dr. Harrison B. Rhodes	27	Compendium of 11 Articles by Dr. Gerald R. Chase and Dr. Harrison B. Rhodes.
II-40	20/08/81	Dr. Jacques Dunnigan	28	Compendium of 13 Articles for Dr. Jacques Dunnigan.
II-41	21/08/81	Dr. Graham W. Gibbs	29	Compendium of 35 Articles by Dr. Graham W. Gibbs.
II-42	27/08/81	Dr. Alison D. McDonald	31	Compendium of 16 Articles by Dr. Alison D. McDonald.
II-43	23/10/81	Dr. John M. Dement	32	Compendium of 12 Articles by Dr. John M. Dement.
II-44	23/10/81	Dr. John M. Dement	32	Excerpt from U.S. National Institute for Occupational Safety and Health. *Criteria for a Recommended Standard — Occupational Exposure to Asbestos*, Tables XI, XII, XXVI. Washington, D.C.: U.S. Department of Health, Education and Welfare, 1972.

II-45	23/10/81	Dr. John M. Dement	32	Excerpt from Lemen, Richard A.; Dement, John M.; and Seiter, Robert E. "Feasibility of Industrial Compliance with New Asbestos Standards," pp. 4, 5 plus assorted tables. Cincinnati, Ohio, NIOSH, n.d. Draft.
II-46	23/10/81	Dr. John M. Dement	32	Kaplan, Samuel D., and Gaffey, William R. "Miners Exposed to Amphibole Mineral: A Retrospective Cohort Mortality Study." Cincinnati, Ohio, U.S. Department of Health and Human Services, June 1981. (Mimeographed.)
II-47	15/01/82	Dr. John M.G. Davis	34	Compendium of 26 Articles by Dr. John M.G. Davis.
II-48	18/02/82	Mr. James F. Reis	35(A)	Assorted materials published by the Asbestos Information Association/North America.
II-49	18/02/82	Mr. James F. Reis	35(A)	Castleman, Barry. "Double Standards: Asbestos in India." *New Scientist* (26 February 1981): 522–523.
II-50	19/02/82	Mr. James F. Reis	35(B)	Letter from Mr. James F. Reis, Director, Asbestos Policy, Johns-Manville Corporation to the Royal Commission on Asbestos, 16 February 1982.
II-51	20/05/82	Dr. Bailus Walker, Jr.	36	Compendium of 20 Articles for Dr. Bailus Walker, Jr.
II-52	28/05/82	Mr. Gunnar Danielson Mr. Arne Westlin Mr. Lars Ettarp Mr. Harald Linton	37	Compendium of 15 Articles for Representatives from Government of Sweden.

Exhibit Number	Date Filed	During Testimony of:	Transcript Volume Number	Exhibit
II-53	01/06/82	Mr. William J. Simpson	38	Compendium of 15 Articles for Mr. William J. Simpson.
II-54(a)	08/06/82	Dr. Jerome J. Vingilis	39	American Medical Association. Committee on Rating of Mental and Physical Impairment. *Guides to the Evaluation of Permanent Impairment*. Chicago: AMA, 1971. Foreword, Preface, and chap. 4.
II-54(b)	08/06/82	Dr. Jerome J. Vingilis	39	Assorted Forms.
II-55	15/06/82	Mr. Robert Sass	40(A)	Compendium of 24 Articles for Mr. Robert Sass.
II-56	15/06/82	Mr. Robert Wilson	40(B)	Compendium of 8 Articles for Mr. Robert Wilson.
II-57	16/08/82	Mr. Ross Hunt	41	Compendium of 8 Articles by Mr. Ross Hunt.
II-58	21/06/82	Mr. Gyan S. Rajhans	42(A)	Compendium of 17 Articles for Mr. Gyan S. Rajhans.
II-59	22/06/82	Mr. Walter Melinyshyn	43(A)	Opening statement before the Royal Commission on Asbestos.
II-60(a)	22/06/82	Mr. Walter Melinyshyn	43(A)	Ontario. Ministry of Labour. Occupational Health and Safety Division. Industrial Health and Safety Branch. *Operations Manual*, section 7.4. "Potential Prosecutions." [Toronto]: revised February 1982; ibid., section 9.2.4. "Forms — Special Action Request." revised March 1980.

II-60(b)	22/06/82	Mr. Walter Melinyshyn	43(A)	Ontario. Ministry of Labour. Occupational Health and Safety Division. Industrial Health and Safety Branch. *Operations Manual.* [Toronto: looseleaf.] Ontario. Ministry of Labour. Construction Health and Safety Branch. *Operations Manual.* [Toronto: looseleaf.]
II-60(c)	22/06/82	Mr. Walter Melinyshyn	43(A)	Ontario. Ministry of Labour. Memorandum from Mr. Paul Hess, Director, Legal Services Branch to Mr. Alan Heath, Director, Standards and Programs Branch, 22 April 1982.
II-61	22/06/82	Mr. James McNair	43(B)	Ontario. Ministry of Labour. Plant Visit Report on Bendix Eclipse of Canada Limited, 14 April 1966.
II-62	22/06/82	Mr. James McNair	43(B)	Ontario. Ministry of Labour. Field Visit Report on Bendix Eclipse Co. Ltd., 10 March 1970.
II-63	22/06/82	Mr. James McNair	43(B)	Ontario. Ministry of Labour. Field Visit Report on Hayes-Dana, Victor Products Division, 21 January 1981.
II-64	23/06/82	Mr. Jack P. Cashman	44	Johns-Manville. Organizational Charts.
II-65	23/06/82	Mr. Jack P. Cashman	44	Johns-Manville. Assorted Bulletins.
II-66	23/06/82	Mr. Jack P. Cashman	44	Manville Corporation. *Manville* [Annual Report 1981]; Manville Canada Inc. *Manville: 1981 In Review.*
II-67(a)	28/06/82	Mr. Bruce C. Machin	45(A)	Johns-Manville. Internal Correspondence from Mr. K.H. Reeve, Toronto Plant to Mr. J.R. Ariss, CPDHQ, Etobicoke, 24 December 1980

Exhibit Number	Date Filed	During Testimony of:	Transcript Volume Number	Exhibit
II-67(b)	28/06/82	Mr. Bruce C. Machin	45(A)	Johns-Manville, Scarborough. Data on Capital Expenditures, 1960–1978.
II-68	28/06/82	Mr. Bruce C. Machin	45(A)	Manville. Internal Correspondence from Mr. R. Dennis Stevens, Toronto Plant to Mr. Bruce C. Machin, West Mall, 23 June 1982.
II-69	28/06/82	Mr. Bruce C. Machin	45(A)	Borough of Scarborough. Board of Health. Resolution on Asbestos, 7 March 1975.
II-70	28/06/82	Mr. Bruce C. Machin	45(A)	Johns-Manville. Toronto Plant. Notice to all Employees. "Smoking and Asbestos." 29 May 1975.
II-71	28/06/82	Mr. Eugene Girdauskas	45(B)	Compendium of 1 Arti[illegible] by Mr. Eugene Girdauskas.
II-72	28/06/82	Mr. Eugene Girdauskas	45(B)	Ontario. Ministry of Labour. Industrial Health and Safety Branch. Inspection Report for Ontario Hydro, 4 June 1980.
II-73	28/06/82	Mr. Eugene Girdauskas	45(B)	Safety Audit, #8 Unit, done by E. Girdauskas and R. Seaton, Members of Health and Safety Committee of the Hearn Generating Station, 26 May 1982.
II-74	29/06/82	Dr. Paul Kotin	46	Compendium of 14 Articles for Dr. Paul Kotin.
II-75	29/06/82	Dr. Paul Kotin	46	Correspondence between Johns-Manville and Dr. Murray M. Finkelstein, Ontario Ministry of Labour.

II-76	30/06/82	Mr. Arthur L. Gladstone	47(A)	Compendium of 8 Articles for Mr. Arthur L. Gladstone.
II-77	10/08/82	Dr. E. Keith Fitzgerald	54	Compendium of 1 Article by Dr. E. Keith Fitzgerald.
IV-1	13/07/82	Mr. John F. McDonald	48	The Workmen's Compensation Board, Ontario. Assorted Policy Statements and Forms.
IV-2	13/07/82	Mr. John F. McDonald	48	The Workmen's Compensation Board, Ontario. Organizational Charts.
IV-3	13/07/82	Mr. John F. McDonald	48	The Workmen's Compensation Board, Ontario. Claims Services Division. *Board Policies and Administrative Directives*, Directives 1–4 on section 42(5) [now section 43(5)] of the *Workers' Compensation Act*.
IV-4	15/07/82	Dr. Charles Stewart	50	Letter from Dr. William J. McCracken, Executive Director, Medical Services Division, The Workmen's Compensation Board, Ontario to Ms. Marion Endicott, Community Legal Worker, Injured Workers' Consultants, 4 March 1982.
IV-5	11/08/82	Mr. Alan G. MacDonald	55	The Workmen's Compensation Board, Ontario. Inter-Divisional Communication from Mr. John C. Neal, Board Actuary to Mr. Alan G. MacDonald, Vice-Chairman of Administration and General Manager, 9 July 1982.

Exhibit Number	Date Filed	During Testimony of:	Transcript Volume Number	Exhibit
IV-6	11/08/82	Mr. Alan G. MacDonald	55	The Workmen's Compensation Board, Ontario. *Experience Rating Plan for Employers*. Toronto: WCB, 1981.
IV-7	12/08/82	Mr. William D. Pearce	56	The Workmen's Compensation Board, Ontario. Data re Special Rehabilitation Assistance Programme, 26 March 1982.
IV-8	12/08/82	Mr. William D. Pearce	56	The Workmen's Compensation Board, Ontario. Data re Special Rehabilitation Assistance Programme, 1 January 1981 - 10 August 1982; 31 December 1980; and 31 December 1979.
IV-9	12/08/82	Mr. William D. Pearce	56	The Workmen's Compensation Board, Ontario. Inter-Divisional Communication from Dr. Douglas W. Dyer, Chest Disease Specialist to Mr. Alex Joma, Secretary, 10 August 1982.

Appendix D

Written Submissions Filed with the Royal Commission on Asbestos

Written Submission Number	Submitted By:
1	Mr. Victor Delmonte
2	Mr. Mark S. Rudolph
3	Borough of Scarborough, Building Department
4	Ms. Lorraine A. Cass
5	Mr. Howard B. Newcombe
6	Mrs. Margaret Moir
7	International Association of Bridge, Structural and Ornamental Iron Workers, Local 721
8	Asbestos Information Association/North America (AIA/NA)
9	R.T. Vanderbilt Company, Inc.
10	Ontario Medical Association, Committee on Public Health
11	American Industrial Hygiene Association, Ontario Local Section
12	Hamilton Labour Council, Hamilton Area Occupational Health and Safety Committee
13	Labour Council of Metropolitan Toronto
14	London and St. Thomas District Labour Councils, Health and Safety Committees

Written Submission Number	Submitted By:
15	Mrs. Frances Day
16	Quebec Asbestos Mining Association
17	Consumers' Association of Canada (Ontario)
18	Johns-Manville Canada Inc.
19	Gouvernement du Québec, Ministère de l'Energie et des Ressources (Mines)
20	Toronto Occupational Health Resource Committee
21	Dow Chemical of Canada, Limited
22	The Board of Education for the City of Toronto
23	Canadian Union of Public Employees (CUPE), Ontario Educational Institutes Co-ordinating Committee
24	Canadian Union of Public Employees (CUPE), Metropolitan Toronto District Council
25	Communications Workers of Canada, Ontario Region
26	Asbestos Victims of Ontario
27	Mr. Frank E. Stokes
28	Ontario Public Service Employees Union (OPSEU)
29	City of Toronto, Department of Public Health
30	Canadian Union of Public Employees (CUPE), Ontario Division
31	Ontario New Democratic Party Caucus
32	Energy and Chemical Workers Union (ECW)
33	Construction Safety Association of Ontario (CSA)
34	International Union, United Automobile, Aerospace and Agricultural Implement Workers of America (UAW), Canadian Region
35	Ontario Federation of Labour (OFL)
36	United Electrical, Radio and Machine Workers of America (UE), National Office
37	Mrs. Odette Dodds
38	The Ontario Association of School Business Officials

Written Submission Number	**Submitted By:**
39	Stelco Inc.
40	Canadian Union of Public Employees (CUPE), Local 27; and Windsor Occupational Safety and Health Council (WOSH)
41	Ms. Helen R. Hindle
42	United Steelworkers of America, National Office, Health and Safety Department
43	Ontario Ministry of Labour
44	Canadian Centre for Occupational Health and Safety
45	Canadian Environmental Law Association
46	Mr. Terry Howes
47	Mrs. Odette Dodds
48	Mr. Dwight Oland
49	Professor Edward J. Farkas
50	Woodsreef Minerals Ltd.
51	Mr. Douglas Wray
52	Mr. D.W. Bishop
53	Provincial Building and Construction Trades Council of Ontario
54	Mr. Bruce B. Koffler
55	Mrs. Marjorie Shorting
56	Ms. Sandra Elia
57	Ontario Hospital Association (OHA)
58	A.O. Wilson Process Equipment Limited
59	The London CUPE Council
60	Bendix Automotive of Canada, Ltd.
61	Ontario Ministry of Labour
62	Professor Frank C. Innes
63	International Association of Heat and Frost Insulators and Asbestos Workers, Insulators Health Hazards Program

Written Submission Number	Submitted By:
64	Mrs. Betty Glaser
65	The Bendix Corporation
66	Hamilton Labour Council, Hamilton Area Occupational Health and Safety Committee
67	Mr. Albert Lever
68	Mr. Wolfgang Palleske
69	The Workmen's Compensation Board, Ontario
70	Mr. G.W. Cousineau
71	Mr. Floyd Lefebvre
72	Mr. R.B. Diamond
73	Mrs. Betty Glaser
74	Mr. Dwight Oland
75	Mrs. Odette Dodds
76	Canadian Union of Operating Engineers and General Workers, Local 110
77	Energy and Chemical Workers Union
78	Ontario Federation of Labour (OFL)
79	United Electrical, Radio and Machine Workers of America (UE), National Office
80	Johns-Manville Canada Inc.
81	Mrs. G.W. Herron
82	Injured Workers' Consultants
83	Mr. Joseph Pagnello
84	Asbestos Victims of Ontario
85	Ontario Ministry of the Environment
86	Ms. Morag Perkins
87	Transport and General Workers Union, U.K.
88	International Association of Heat and Frost Insulators and Asbestos Workers Union, Local 95, Health and Safety Committee

Appendix E

Oral Submissions Before the Royal Commission on Asbestos

Date	Submission	Transcript Volume Number:
16 February 1981	Ontario Federation of Labour	1
16 February 1981	Labour Council of Metropolitan Toronto	1
16 February 1981	Energy and Chemical Workers Union	1
16 February 1981	Communications Workers of Canada, Ontario Region	1
16 February 1981	Canadian Centre for Occupational Health and Safety	1
16 February 1981	Asbestos Victims of Ontario	1
16 February 1981	Mrs. Odette Dodds	1
17 February 1981	Quebec Asbestos Mining Association	2
17 February 1981	Mr. Terry Howes	2
17 February 1981	Ontario New Democratic Party Caucus	2
17 February 1981	Johns-Manville Corporation	2
17 February 1981	International Union, United Automobile, Aerospace and Agricultural Implement Workers of America, Canadian Region	2
17 February 1981	Canadian Environmental Law Association	2
18 February 1981	International Association of Bridge, Structural and Ornamental Iron Workers, Local 721	3
18 February 1981	Construction Safety Association of Ontario	3

Date	Submission	Transcript Volume Number:
18 February 1981	Ontario Federation of Labour	3
18 February 1981	American Industrial Hygiene Association, Ontario Local Section	3
18 February 1981	Injured Workers' Consultants	3
18 February 1981	Toronto Occupational Health Resource Committee	3
18 February 1981	City of Toronto, Board of Education	3
18 February 1981	Borough of Scarborough, Building Department	3
18 February 1981	City of Toronto, Department of Public Health, Health Advocacy Unit; and Food Control and Sanitation Division	3
18 February 1981	Canadian Union of Public Employees, Ontario Division	3
18 February 1981	Canadian Union of Public Employees, Ontario Educational Institutes Co-ordinating Committee; and Metropolitan Toronto District Council	3
18 February 1981	Ontario Public Service Employees Union	3
19 February 1981	The Ontario Association of School Business Officials	4
19 February 1981	Asbestos Information Association/North America	4
19 February 1981	Ontario Ministry of Labour	4
19 February 1981	Woodsreef Minerals Ltd.	4
19 February 1981	United Steelworkers of America, National Office, Health and Safety Department; and District 6	4
20 February 1981	Asbestos Victims of Ontario	5
20 February 1981	United Electrical, Radio and Machine Workers of America, National Office	5
20 February 1981	Mr. Alan Davis	5
27 March 1981	Canadian Union of Public Employees, Local 27; and Windsor Occupational Safety and Health Council	6
27 March 1981	Professor Frank C. Innes	6
27 March 1981	Ontario Ministry of Labour	6

Date	Submission	Transcript Volume Number:
27 March 1981	International Union, United Automobile, Aerospace and Agricultural Implement Workers of America, Canadian Region; and Local 195	6
27 March 1981	London and District Labour Council; and St. Thomas and District Labour Council	6
27 March 1981	Canadian Union of Public Employees, London District Council	6
8 June 1981	Provincial Building and Construction Trades Council of Ontario	7
8 June 1981	Gouvernement du Québec, Ministère de l'Energie et des Ressources	7
8 June 1981	International Association of Heat and Frost Insulators and Asbestos Workers	7
8 June 1981	Dow Chemical of Canada, Limited	7
8 June 1981	Mrs. Betty Glaser	7
5 July 1982	Mrs. Betty Glaser	47(B)
5 July 1982	Mrs. Lilian Perkins and Ms. Morag Perkins	47(B)
5 July 1982	Mrs. Odette Dodds	47(B)
5 July 1982	Mr. Joseph Pagnello	47(B)
5 July 1982	Mrs. Camille Haineault	47(B)
5 July 1982	Mr. Ed Cauchi	47(B)
5 July 1982	International Association of Heat and Frost Insulators and Asbestos Workers, Local 95	47(B)
5 July 1982	Energy and Chemical Workers Union	47(B)
5 July 1982	Mr. Floyd Lefebvre	47(B)
28 January 1983	Asbestos Victims of Ontario	58
28 January 1983	Energy and Chemical Workers Union	58
28 January 1983	Injured Workers' Consultants	58
28 January 1983	Ontario Federation of Labour	58
28 January 1983	Asbestos Information Association/North America	58
28 January 1983	Quebec Asbestos Mining Association	58

Date	Submission	Transcript Volume Number:
28 January 1983	Government of Ontario	58
28 January 1983	Mr. Ed Cauchi	58
28 January 1983	Mrs. Odette Dodds	58

Appendix F

Publications and Other Materials Released by the Royal Commission on Asbestos

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no. 2. *Workers' Compensation and Asbestos in Ontario*, by Peter S. Barth. 1982.
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no. 3. *Policy Options in the Regulation of Asbestos-Related Health Hazards*, by Carolyn J. Tuohy and Michael J. Trebilcock. 1982.
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no. 5. *Living with Contradictions: Health and Safety Regulation and Implementation in Ontario*, by G. Bruce Doern, Michael Prince, and Garth McNaughton. 1982.
ISBN 0-7743-7056-4 $10.00

no. 6. *Worker Attitudes About Health and Safety in Three Asbestos Brake Manufacturing Plants*, by Sally Luce and Gene Swimmer. 1982.
ISBN 0-7743-7057-2 $5.00

no. 7. *The Technical Feasibility and Cost of Controlling Workplace Exposure to Asbestos Fibres*, by Gordon M. Bragg. 1982.
ISBN 0-7743-7311-3 $5.50

no. 8. *Asbestos in Buildings*, by Donald J. Pinchin. 1982.
ISBN 0-7743-7323-7 $10.00

Note: *ISSN for Study Series: 0714-0169.

no. 9. *Measurement of Asbestos Fibre Concentrations in Workplace Atmospheres*, by Eric J. Chatfield. 1982.
ISBN 0-7743-7777-1 $2.75

no. 10. *Measurement of Asbestos Fibre Concentrations in Ambient Atmospheres*, by Eric J. Chatfield. 1983.
ISBN 0-7743-8175-2 $3.25

Other Publications

Proceedings of The Royal Commission on Asbestos, First Public Meeting, Friday, October 31, 1980. Chairman: J. Stefan Dupré. Reporter: Elizabeth J. Hiscott, B.Sc. 1980.
ISBN 0-7743-6024-0 $1.50

Proceedings of The Royal Commission on Asbestos, Second Public Meeting, Friday, December 12, 1980. Chairman: J. Stefan Dupré. Reporter: Lydia Dotto. 1981.
ISBN 0-7743-6086-0 $1.50

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Background Paper Series

no. 1. *A Survey of Asbestos Policies in Canada with Particular Emphasis on Ontario*, by Sandra Glasbeek. 1981.

no. 2. *A Review of Four Major Reports on the Health Hazards of Asbestos*, by The Canadian Centre for Occupational Health and Safety. 1981.

Brochure and Newsletters

Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario, brochure outlining Terms of Reference. 1980.
ISBN 0-7743-5474-7

no. 80-1, November 1980
no. 81-1, February 1981
no. 81-2, March 1981
no. 81-3, April 1981
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Reviews of Studies Prepared for the Royal Commission on Asbestos

	Reviewer	Author(s) of Study	Title of Study
1.	Steven Kelman	G. Bruce Doern	*The Politics of Risk: The Identification of Toxic and Other Hazardous Substances in Canada*
2.	Steven Kelman	G. Bruce Doern Michael Prince Garth McNaughton	*Living with Contradictions: Health and Safety Regulation and Implementation in Ontario*
3.	Harry Arthurs	Morley Gunderson Katherine Swinton	*Collective Bargaining and Asbestos Dangers at the Workplace*
4.	Gordon B. Milling	Morley Gunderson Katherine Swinton	*Collective Bargaining and Asbestos Dangers at the Workplace*
5.	Lester Lave	Morley Gunderson Katherine Swinton	*Collective Bargaining and Asbestos Dangers at the Workplace*
6.	Lester Lave	Carolyn J. Tuohy Michael J. Trebilcock	*Policy Options in the Regulation of Asbestos-Related Health Hazards*
7.	Adam S. Little	Peter S. Barth	*Workers' Compensation and Asbestos in Ontario*
8.	Terence G. Ison	Peter S. Barth	*Workers' Compensation and Asbestos in Ontario*
9.	Richard Merrill	G. Bruce Doern	*The Politics of Risk: The Identification of Toxic and Other Hazardous Substances in Canada*
10.	Linda Jolley	Carolyn J. Tuohy Michael J. Trebilcock	*Policy Options in the Regulation of Asbestos-Related Health Hazards*
11.	Linda Jolley	G. Bruce Doern Michael Prince Garth McNaughton	*Living with Contradictions: Health and Safety Regulation and Implementation in Ontario*
12.	Nicholas Ashford	Gordon M. Bragg	*The Technical Feasibility and Cost of Controlling Workplace Exposure to Abestos Fibres*

Appendix G

Research, Administrative, and Secretarial Staff of the Royal Commission on Asbestos

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Edwina Macht

Appendix H

Consultants Engaged by the Royal Commission on Asbestos

Name	Institutional Affiliation
David Andrews	Department of Preventive Medicine and Biostatistics, University of Toronto
Peter S. Barth	Department of Economics, University of Connecticut
David V. Bates	Department of Medicine, University of British Columbia
Gordon M. Bragg	Department of Mechanical Engineering, University of Waterloo
Eric J. Chatfield	Department of Applied Physics, Ontario Research Foundation
G. Bruce Doern	School of Public Administration, Carleton University
Emmanuel Farber	Faculty of Medicine, University of Toronto
Morley Gunderson	Centre for Industrial Relations, University of Toronto
John Jaworski	National Research Council Canada
Richard Kuntze	Special Programmes, Ontario Research Foundation
David C.F. Muir	Occupational Health Program, McMaster University
Julian Peto	Imperial Cancer Research Fund, University of Oxford
Donald J. Pinchin	D.J. Pinchin Technical Consulting Ltd.
J. Robert S. Prichard	Faculty of Law, University of Toronto
Samuel A. Rea, Jr.	Institute for Policy Analysis, University of Toronto
Robin S. Roberts	Department of Clinical Epidemiology and Biostatistics, McMaster University
Neil Rowlands	Department of Mining and Metallurgical Engineering, McGill University
Harry Shannon	Occupational Health Program, McMaster University
John Steel	Nuffield Department of Industrial Health, University of Newcastle-upon-Tyne
John L. Sullivan	Department of Chemical Engineering, University of Western Ontario

Name	Institutional Affiliation
Katherine Swinton	Faculty of Law, University of Toronto
Michael J. Trebilcock	Faculty of Law, University of Toronto
Carolyn J. Tuohy	Department of Political Science, University of Toronto
Edwin Warrick	Airport Facilities Branch, Transport Canada
Fred J. Wicks	Department of Mineralogy, Royal Ontario Museum
Albert Winer	Formerly with CANMET, Energy, Mines and Resources Canada

Biographical Notes on Commissioners

J. Stefan Dupré, Ph.D., D.Sc.Soc., LL.D., D.U., is a professor of political science at the University of Toronto, and a past Chairman of the University's Department of Political Economy. He served as founding Chairman of the Ontario Council on University Affairs. From 1969 to 1977, Dr. Dupré was a member of the National Research Council of Canada. Between 1977 and 1980, he was a member of the Professional Organizations Committee of the Attorney General of Ontario. He is a Past President of the Institute of Public Administration of Canada.

J. Fraser Mustard, M.D., Ph.D., D.Sc., F.R.C.P.(C), F.R.S.C., is President of the Canadian Institute for Advanced Research. He is also a professor of pathology at McMaster University in Hamilton, Ontario, where he was Dean and/or Vice-President of Health Sciences from 1972 to 1982. His career has spanned the fields of academia, administration, and research with special interest in the factors and mechanisms causing cardiovascular disease. From 1966 to 1972, Dr. Mustard was a member of the Ontario Council of Health. In 1973–1974, he was Chairman of the Task Force on Health Planning for the Ministry of Health. From 1973 to 1975, he chaired the Committee on Diet and Cardiovascular Disease for Health and Welfare Canada. Between 1974 and 1981, he served as a member of the Ontario Council on University Affairs. He was Chairman of the Minister of Labour's Advisory Council on Occupational Health and Occupational Safety from 1978 to 1984.

Robert J. Uffen, O.C., P.Eng., Ph.D., D.Sc., F.R.S.C., is the past Dean of the Faculty of Applied Sciences at Queen's University in Kingston, Ontario. In the summer of 1980, Dr. Uffen resumed activities as a professor of engineering and geophysics at Queen's Department of Geology. He is a fellow of the Royal Society of Canada and a former member of the Science Council of Canada. He is a fellow of the Geological Society of America and, before joining the Faculty at Queen's, he acted as the Chief Science Advisor to the federal Cabinet. He was a member of the National Research Council of Canada's Associate Committee on Scientific Criteria for Environmental Quality. In 1983, Dr. Uffen was made an Officer of the Order of Canada.

Selected Bibliography

Selected Bibliography*

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In this Selected Bibliography, most cross-references are in the format of "Exhibit X, Tab Y." In these instances, "Exhibit X" refers to a collection of articles or other materials written by, or relevant to, a particular witness; these were bound and given a collective exhibit number during our hearings. "Tab Y" refers to an index number assigned to each article within the Exhibit. This format became a shorthand way of referring to articles as we were discussing them during the testimony of witnesses.

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